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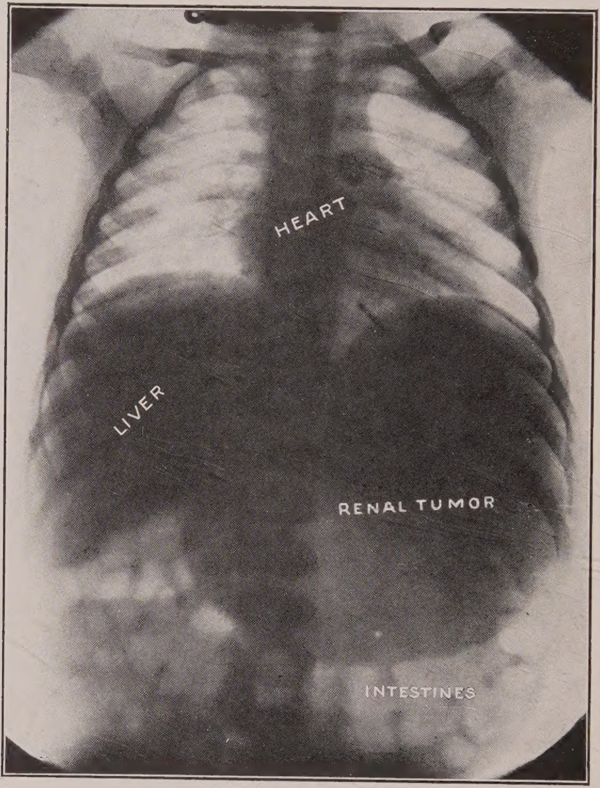

Radiograph of a Child's Thorax and Abdomen. The Heart, Liver and a Renal Neoplasm Are Made Visible by the Air around Them in the Lungs and Intestines.

(*Frontispiece*)

PHYSICAL DIAGNOSIS

BY

RICHARD C. CABOT, M. D.

PROFESSOR OF MEDICINE IN HARVARD UNIVERSITY
FORMERLY CHIEF OF THE WEST MEDICAL SERVICE
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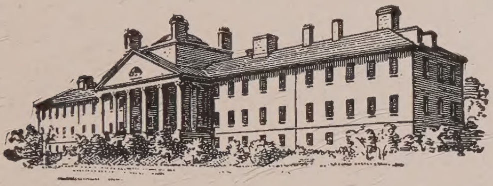

MASSACHUSETTS GENERAL HOSPITAL

Eighth Edition

REVISED AND ENLARGED, WITH SIX PLATES
AND 279 FIGURES IN THE TEXT

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TO

FREDERICK C. SHATTUCK, M. D.

FORMERLY JACKSON PROFESSOR OF CLINICAL MEDICINE
IN HARVARD UNIVERSITY

IN EVIDENCE OF MY APPRECIATION OF
THE EXAMPLE OF SINCERITY, COMMON SENSE, AND ENTHUSIASM
ESTABLISHED BY HIM IN THE TEACHING AND
THE PRACTICE OF MEDICINE

PREFACE TO THE EIGHTH EDITION

The number of changes since the last edition has been so great as to require a complete resetting of the book. The section on emphysema has been radically changed. The chapter on electro-cardiograph has, with Dr. Paul D. White's help, been brought up to date. I wish to thank him here for expert and generous aid.

The increasing value of x-ray in the diagnosis especially of pulmonary and genito urinary diseases has led me to add many additional instructions and many new sentences. A number of new X-ray pictures have been inserted. For them I am indebted to Dr. George W. Holmes and his associates in the X-ray department of the Mass. General Hospital.

R. C. C.

August, 1923

PREFACE

This book endeavors to present an account of the diagnostic methods and processes needed by competent practitioners of the present date. It differs from other books on the subject in that it makes no attempt to describe technical processes with which the writer has no personal familiarity and gives no space to the description of tests which he believes to be useless.

To gain genuine familiarity with all the technical processes described in most books on physical diagnosis—such familiarity as makes one competent to use them with due regard for the sources and limits of error inherent in them—needs more than the life-time of one man. But unless one has one's self used a technical process long enough to gain this sort of mastery over it, one cannot properly describe it, far less recommend it to others. Because of my lack of personal acquaintance with such methods as cystoscopy, ophthalmoscopy, and laryngoscopy I have attempted no description of them, although I believe they should sooner or later be mastered by every internist. All that I have described I know by prolonged use.

A book constructed on this basis should make obvious what its writer considers important and what unimportant, and reveal therein not only his opinions but his personal limitations. But I believe there is no longer a demand for books that attempt impartially to present all that has been or is now thought of value by some one. The personal equation cannot and should not be ignored. In diagnosis as in therapeutics "*What do you find valuable?*" is the question that our contemporaries ask of any one of us, not "What has been recommended?"

In the endeavor further to break down the false distinction between clinical diagnosis and laboratory diagnosis I have described all the methods of getting at an organ—*e.g.*, the kidney—in a single section. Palpation, thermometry, urinalysis are different processes by which we may gather information about the kidney. The student should be accustomed to think of them and practice them in close sequence.

For the same reason the most important methods of investigating the stomach have been grouped together without any distinction of "clinical" and "laboratory" procedure.

For the illustrations I owe many thanks to many persons, especially to Drs. Frank Billings, A. E. Boycott, E. H. Bradford, E. R. Carson, J. Everett Dutton, R. T. Edes, Joel E. Goldthwait, J. S. Haldane, Frederick T. Lord, R. W. Lovett, H. C. Masland, S. J. Meltzer, Percy Musgrave, R. F. O'Neil, J. E. Schadle, William H. Smith, W. S. Thayer, and G. L. Walton; also to the editors of *The Boston Medical and Surgical Journal*, *The St. Paul Medical Journal*, *American Medicine*, *The Journal of Experimental Medicine*, and *The Lancet*.

My assistant, Dr. Mary W. Rowley, has helped me very much with the index as well as with other parts of the book.

190 MARLBORO ST., BOSTON.
June, 1905.

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PHYSICAL DIAGNOSIS

CHAPTER I

DATA RELATING TO THE BODY AS A WHOLE

I. Weight

To weigh the patient should be part of every physical examination, and every physician's office should contain a good set of scales.

1. *Gain in weight*, aside from seasonal changes, the increase in normal growth, and convalescence from wasting diseases, means usually:

(*a*) Obesity.

(*b*) The accumulation of serous fluid in the body—dropsy, evident or latent.

The first of these needs no comment. *Latent* accumulation of fluid, not evident in the subcutaneous tissues or serous spaces, occurs in some forms of uncompensated cardiac or renal disease and in diabetes, and gives rise to an increase in weight which may delude the physician with the false hope of an improvement in the patient's condition, but in reality calls for derivative treatment (diuresis, sweating).

Obvious dropsy has, of course, the same effect on the weight and the same significance.

(*c*) Myxœdema is a cause of increased weight, *i.e.*, especially when the myxœdematous infiltration is widespread (see below, page 11).

2. *Loss of Weight.*—The aging process is so often associated with loss of weight that some writers speak of the "*cachexia of old age.*" In some, a rapid loss of superfluous fat may occur at moderate age, *e.g.*, at fifty-five, and may give rise to grave apprehension though the general health remains good and no known disease develops.

Aside from this physiological change of later life, most cases of loss of weight are due to:

(*a*) Malnutrition.

(*b*) Loss of sleep (whether from pain or other cause).

(*c*) Infectious fevers and other toxæmic states.

Under the head of *malnutrition* come the cases of œsophageal stricture, chronic dyspepsia (usually with peptic ulcer) and gastric cancer, chronic diarrhœa, the atrophies of infancy, diabetes mellitus, and the rare cases of anorexia nervosa.

Loss of sleep is, I believe, the chief factor in the emaciation occurring in many painful illnesses as well as in various other types of disease. It is only in this way that I can account for the marked emaciation in many cases of thoracic aneurism and of gall-stones.

Toxœmia is, I suppose, accountable for part at least of the emaciation in neoplasms, typhoid, cirrhotic liver, and tuberculosis. It is especially important to suspect tuberculosis and look for it in any patient who has lost weight without any obvious cause, for such a loss is often an early symptom of the disease.

Accelerated or increased metabolism is present in Graves' disease and may be one of the earliest symptoms. Even if the patient takes more than his normal share of food he may lose weight steadily.

II. TEMPERATURE

The method of taking temperature is too familiar to need explanation, but the student should be aware of the fact that hysterics and malingerers can and often do raise the mercury in the bulb by various manœuvres, unless they are vigilantly watched. Dipping the bulb into hot water, shaking the mercury upward toward the higher degrees of the scale, and possibly friction with the tongue (?) are to be suspected.

In comatose or dyspnœic patients and in infancy the temperature is best taken by rectum. In others we must be sure that the lips do not remain open during the test, so as to reduce the temperature of the mouth.

1. *Fever, i.e.*, a temperature above 99° F., has much more diagnostic value in adults than in infancy and childhood. In the latter it is often impossible to make out any pathological condition to account for a fever. After childhood the vast majority of fevers are found to be due to:

(*a*) Infectious disease or inflammation of any type.

(*b*) Toxæmia without infection—a much less common and less satisfactory explanation. Cancer of the liver and Graves' disease are examples.

(*c*) Disturbance of heat regulation—as in sunstroke, after the use of atropine, and in nervous excitement, *e.g.*, just after entering a hospital.[1]

(*d*) After hæmorrhage there may be marked fever for which no cause is clear.

For such causes we search when the thermometer indicates fever.

Types of fever often referred to are:

(*a*) "*Continued fever*," one which does not return to normal at any period in the twenty-four hours, as in many cases of typhoid, pneumonia, and tuberculosis.

(*b*) "*Intermittent*," "*hectic*," or "*septic*" fever, one which disappears once or more in twenty-four hours, as in double tertian malaria and septic fevers of various types (including mixed infections in tuberculosis).

A fever which disappears suddenly and permanently is said to end by "*crisis*," while one which gradually passes off in the course of several days ends by "*lysis*."

Long-continued fevers—*i.e.*, those lasting two weeks or more without obvious cause—are usually due (in the temperate zone) to one of three causes:—*Typhoid*, *tuberculosis*, *sepsis*.

In 1,000 "long fevers" (as above defined) the following causes were found in the medical records of the Massachusetts General Hospital:

Typhoid Fever	586	926, or 92.6 per cent.
Tuberculosis	192	
Pyogenic Infections	148	
Epidemic Meningitis	27	74, or 7.4 per cent.
"Influenza"	10	
Infectious Arthritis ("rheumatism")	9	
Leucæmia	5	
Cancer	4	
Syphilis	2	
Miscellaneous	17	

Since the last 7.4 per cent. here listed represent fevers whose cause is usually obvious, it is substantially true to say that *any long obscure fever arising in the temperature zones is due to typhoid, tuberculosis or sepsis*. Under sepsis I include vegetative endocarditis ("benign" or "malignant"), urinary and biliary infections, all local inflammatory processes and generalized bacterial infections with or without a known portal of entry.

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[1] The latter event may also reduce (temporarily) a high fever to normal or below it. In *coma from any cause* (uræmia, cerebral hemorrhage, diabetic coma) fever often occurs.

2. *Subnormal temperature* is often seen in wasting disease (cancer), nephritis, uncompensated heart disease, and myxœdema. It is rarely of diagnostic value, but is a rough measure of the degree of prostration. It may be present in health.

3. *Chills* (due usually to a *sudden* rise in temperature) are seen chiefly in: (*a*) Sepsis of any type, especially urinary or biliary sepsis;[1] (*b*) Malaria; (*c*) Onset of acute infections; (*d*) "Nervous" states.

After the passage of a catheter, after or during labor, after infusion of saline solution or any serum or antitoxin a chill is often seen, but not easily explained.

True chill, with shivering and chattering teeth, is distinguished from *chilliness* without any shivering. Chilliness is far less significant and often goes without fever; true chill rarely does.

The cause of true chills can usually be determined by blood examination (leucocytosis, malarial parasites) and by the general physical examination. Chills without any abnormal physical signs and with normal blood are most often due to sepsis in the kidney, liver or bile ducts. Fever without explanation is sometimes from dental sepsis or sinus infection.

4. *Night Sweats and Day Sweats.*

Sweating in disease seems to be conditioned by: (*a*) Fever (infection); (*b*) Weakness; (*c*) Sleep.

A phthisical patient who falls asleep in the daytime will sweat then and there, and the sweating will stop when he wakes. In typhoid fever and pneumonia sweating often begins in convalescence when the temperature is nearly or quite normal. In alcoholism, hyperthyroidism, and neurasthenic states we sometimes see sweating without fever. In France pretty much *all* dispensary patients complain of night sweats, perhaps because of their sleeping habits.

Sepsis, acute rheumatism, and tuberculosis are the infections most often accompanied by sweating. In rickets the head sweats especially.

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[1] Staphylococcus or B. coli infections of the kidney and its pelvis, cholangitis with stone in the common bile duct.

CHAPTER II

THE HEAD AND FACE; THE NECK

THE HEAD AND FACE

Almost all that we can learn about the manifestations of disease on the head and face is to be learned by the use of our eyes, by *inspection*, as the term is, and by *x*-ray. Other methods—percussion, palpation—yield but little. I shall begin at the top.

I. The Cranial Vault

1. *The Shape and Size of the Cranium*

The shape and size of the cranium concern us, especially in children.

(*a*) *Abnormally small* crania (microcephalia) are apt to mean idiocy of syphilitic origin, especially if the sutures are closed.

(*b*) An *abnormally large head* is seen in *hydrocephalus* (see Fig. 1), associated with enormous "open" areas uncovered by bone and a peculiar downward inclination of the eyes, which are partly covered by the eyelids and show a white margin above the iris. This condition is to be distinguished from the:

(*c*) *Rachitic head*, which is flatter at the vertex and more protuberant at the frontal eminences, giving it a *squarish* outline, contrasted with the *globular* shape and rounded vertex of the hydrocephalic. In rickets there are no changes in the eyes.

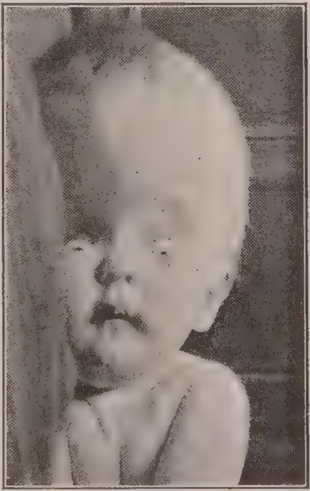


Fig. 1.—Hydrocephalus.

(*d*) In *congenital syphilis* we see a square head, prominent in the forehead, sloping down to a narrow chin.

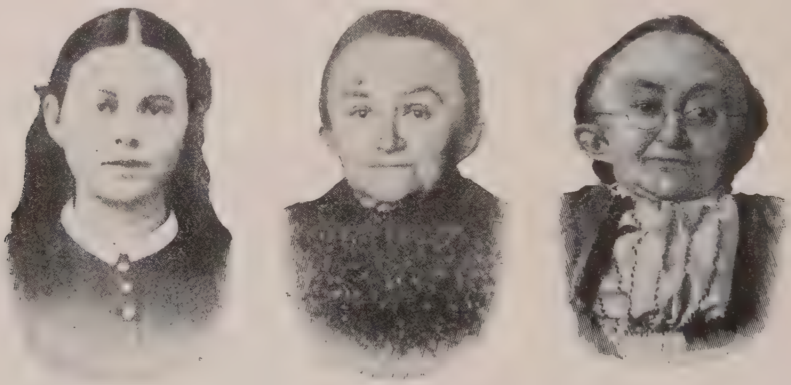
(*e*) In adult life an enlargement of the skull, due to bony thickening, forms part of the rare disease, *osteitis deformans* (Paget's disease), associated with thickening and bowing of the long bones (see Fig. 2).

(*f*) *Myelomata* of the skull may or may not be accompanied by a leukæmic blood and a greenish staining of the tumor tissues. They are recognized by the concurrent presence of the Bence-Jones protein in the urine, by the x-ray, the negative Wassermann reaction and finally the histological examination of an excised node (see Figs. 3 and 4).

Hypernephromata may exhibit a cranial metastasis. With such a tumor the presence of hematuria and enlarged kidney is suggestive.

2. *The Fontanels*

The anterior and larger fontanel remains about the same size for the first year of life, then diminishes, and closes about the twentieth month. The posterior closes in about six weeks. In rickets, hydrocephalus, hereditary syphilis, and cretinism, the fontanels and sutures remain open after the normal time limit.



a *b* *c*

FIG. 2.—Paget's Disease. (Edes.) *a*, Before onset of hyperostosis cranii. *b*, After onset of hyperostosis cranii. *c*, Later still.

(*a*) *Bulging fontanels* mean increased intracranial tension (hydrocephalus, hemorrhage, meningitis, or any acute febrile disease without dyspnœa). (*b*) *Depressed fontanels* are seen in severe diarrhœa, wasting diseases, collapsed states, and acute dyspnœic conditions.

3. *The Hair*

(*a*) *A rachitic child* often rubs the hair off the back of its head by constant rolling on the pillow. (This is associated with profuse sweating of the head.) Patchy baldness occurs in the skin disease *alopecia areata*, and occasionally over the painful area in trigeminal neuralgia.

(*b*) *General loss of hair* occurs normally after many acute fevers and with advancing age. Early baldness (under thirty-five) is often hereditary. *Syphilis* may produce a rapid loss of hair, local or general, and the same is true of *myxœdema;* but in both these diseases the hair usually grows again in convalescence.

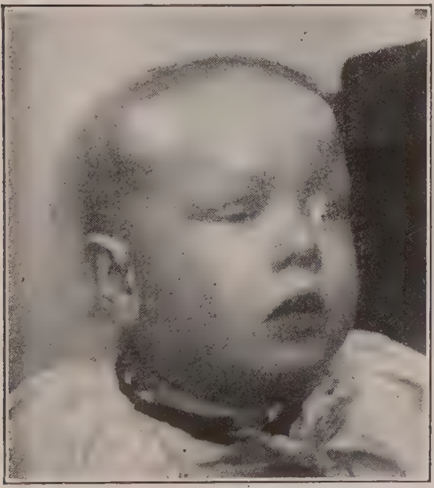


FIG. 3.—Multiple Myelomata.

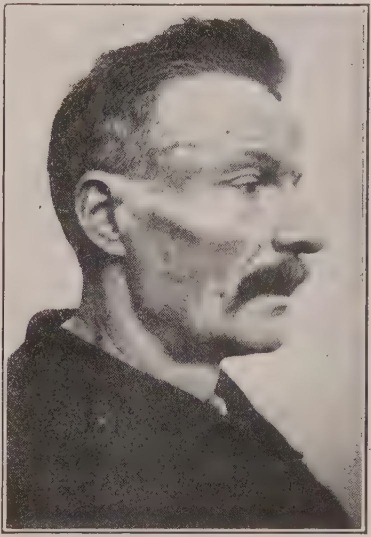


FIG. 4.—Multiple Myelomata.

(*c*) *Parasites* (*pediculi*) are worth looking for in the dirtier classes and those associated with them (teachers). Their eggs adhere to the hairs and are familiarly known as "nits." An eczema or itching dermatitis and an adenitis often results.

II. THE FOREHEAD

Scars, eruptions, and bony nodes are important.

(*a*) *Scars* may be due to trauma or to old syphilitic periostitis. The epileptic often cuts his forehead in falling.

(*b*) *Eruptions* often seen on the forehead are those of acne, syphilis, and smallpox. These may resemble each other closely, and are to be distinguished by the history, the presence of lesions on other parts of the body, and the concomitant signs (fever, prostration, etc.).

(*c*) *Nodes* may be the result of many bumps in childhood or may be caused by a syphilitic periostitis or neoplasms (see Figs. 3, 4, 5 and 7). The history must decide.

(*d*) Evidence of frontal sinusitis may be found (see Fig. 9).

The characteristic history of pain due to frontal sinusitis is that one wakes in the morning free from it, that within an hour or two, and usually at about, the same hour, a milder or severer ache over one eyebrow begins, continues for a few hours, but ordinarily wears off by noon. The regular recurrence and localization are characteristic of sinusitis.

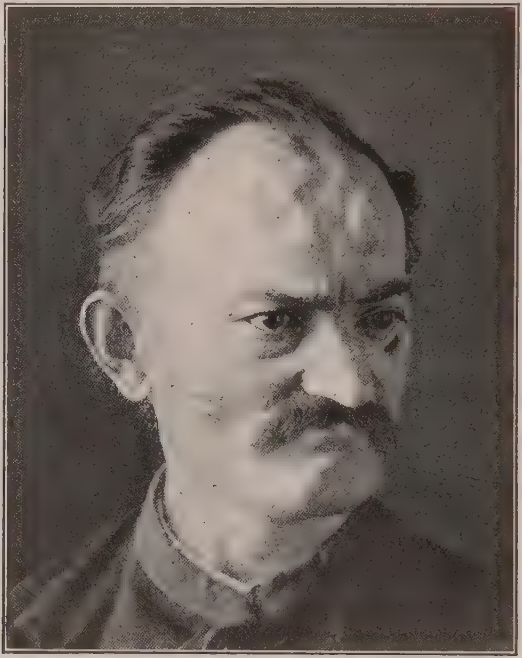


FIG. 5.—Syphilis of the Frontal Bone. (Curschmann.)

III. THE FACE AS A WHOLE

Very characteristic even at a glance is the face of (*a*) *acromegalia*. A strong family likeness seems to pervade all well-marked cases (see Figs. 7 and 8). The huge, bony "whopper jaw" is the most striking item, then the prominent cheek bones, and the ridge above the eyes. The nose and chin are very large.

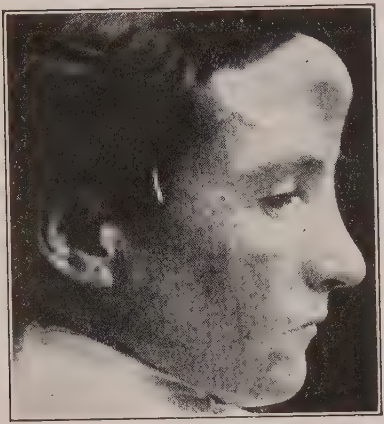


FIG. 6.—Gumma Involving Frontal Bone.

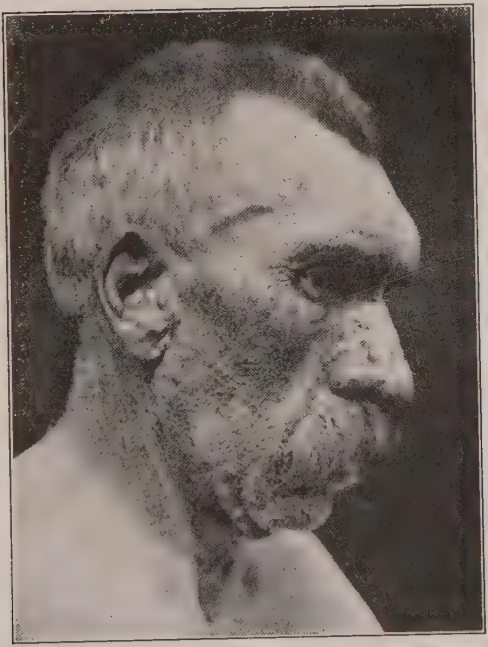


FIG. 7.—Acromegalia.

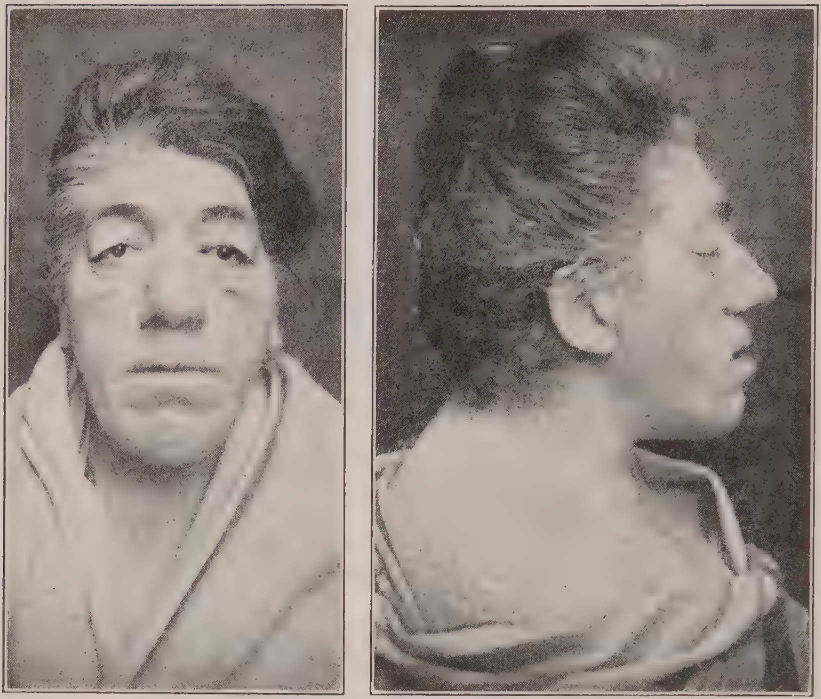


FIG. 8. –Typical Face in Acromegaly.

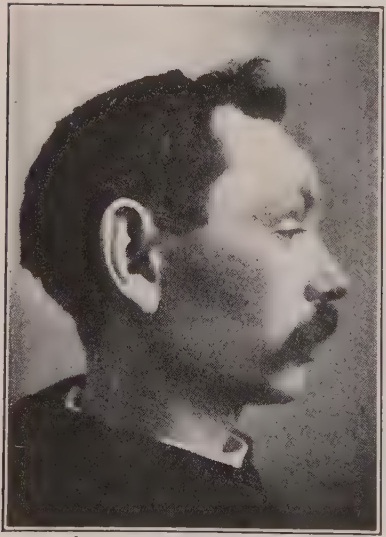


FIG. 9.—Frontal Sinusitis.

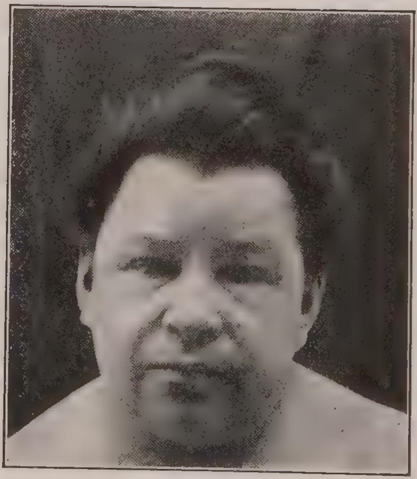
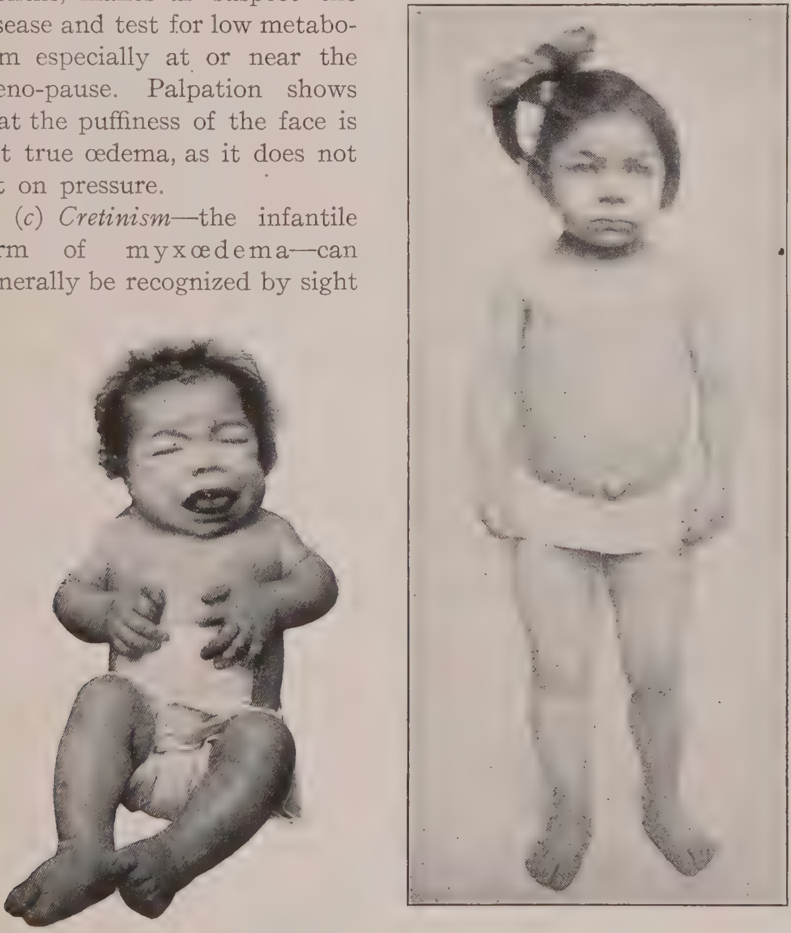


FIG. 10.—Myxœdema.

(*b*) *Myxœdema* (see Fig. 10) is not so characteristic and might easily be mistaken for nephritis or normal stupidity with obesity. The presence of dry skin, falling hair, mental dulness, and subnormal temperature, all supervening simultaneously within a few weeks or months, makes us suspect the disease and test for low metabolism especially at or near the meno-pause. Palpation shows that the puffiness of the face is not true œdema, as it does not pit on pressure.

(*c*) *Cretinism*—the infantile form of myxœdema—can generally be recognized by sight alone (see Figs. 11 and 12). Here the tongue is often protruded, and there are often pot-belly and deformed legs.



FIGS. 11 and 12.—Cretinism.

(*d*) In *adenoids* of the nasopharynx the child's mouth is often open, the nose looks pinched, the expression is stupid (see Fig. 13). There is a history of mouth-breathing and snoring, with frequent "colds," ear troubles and a high-arched palate.

(*e*) In *paralysis agitans* and *encephalitis lethargica* the "*mask-like*" face shows almost no change of expression, whatever the patient says or does. The neck is usually inclined forward, and so rigid that when the patient wishes to look to right or left his whole body rotates like a statue on a pivot. In some cases tremor is absent and the characteristics just mentioned are then of great importance in diagnosis.

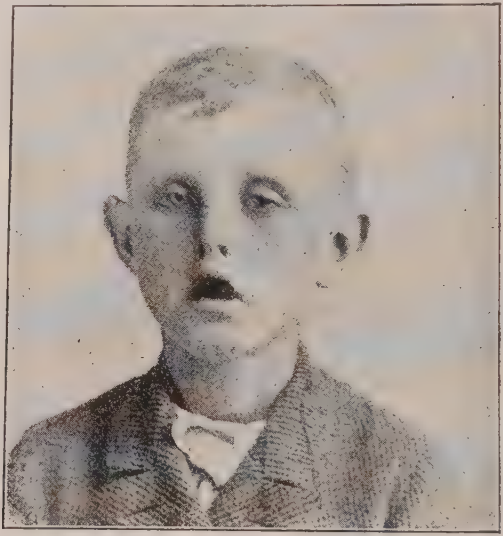


FIG. 13.—Adenoid Face. (Schadle.)

(*f*) In *Graves' disease* (thyrotoxicosis) the *startled* or frightened look is characteristic, though the expression is almost wholly due to the bulging of the eyes and their quick motions (Fig. 14).

(*g*) In *leprosy* the general expression is of a *superabundance of skin* on the patient's face, reminding us of some animal ("leonine face") (Fig. 15).

(*h*) In early *phthisis* one often notices the clear, delicate skin, fine hair, long eyelashes, wide pupils—"appealing eyes." Pallor and a febrile flush (hectic) come later in some cases.

(*i*) *After vomiting* the face has often a drawn, pinched, anxious look, which has often been supposed to be characteristic of general peritonitis, intestinal obstruction, or other diseases accompanied by vomiting; but I can not recognize any single expression as characteristic of peritoneal lesions.

(*j*) *Chronic alcoholism* may be shown not only in a red nose, but oftener in a peculiar, *smoothed-out* look, due, I suppose, to an extra but evenly distributed accumulation of subcutaneous fat.

(*k*) An *œdematous* or swollen face is much more easily noticed by the patient or his friends than by one who is not familiar with his normal look. It usually points to nephritis, but may occur in heart disease, diabetes and sometimes (especially in the morning) without any known cause. When combined with anæmia, the puffy face gives a peculiar "pasty" look (chronic glomerulo-nephritis).

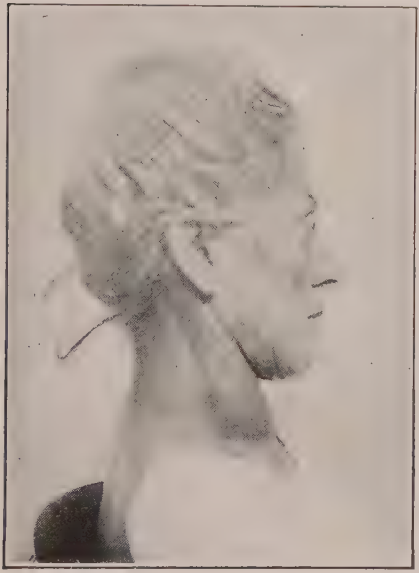


FIG. 14.—Exophthalmic Goitre. (Meltzer.)

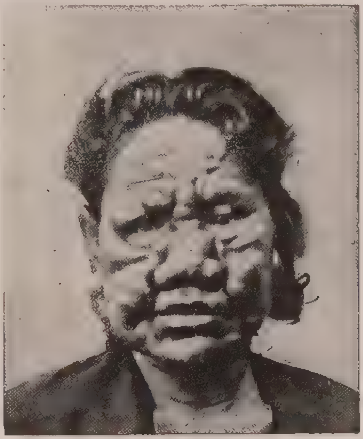


FIG. 15.—Face in Leprosy.

IV. MOVEMENTS OF THE HEAD AND FACE

1. *The Shaking Head*

This occurs often in old age, occasionally in *paralysis agitans* (which oftener affects the hands), and in *toxic* conditions (alcohol tobacco, opium). In some cases no cause can be found.

2. *Spasms of the Face*

Spasms of the face, *i.e.*, sudden, quick contractions of certain facial muscles, such as winking-spasm, jerking of a corner of the mouth, or sniffing, occur chiefly·

(*a*) As a matter of *habit* without other disease. This is *chronic.*

(*b*) As a part of the acute infectious disease *chorea*, with similar "restless" motions of the hands and feet. We often see these spasms in school-children; occasionally in pregnant women. The disease is probably due to the same streptococcus which produces (simultaneously or at other times) the youthful types of polyarthritis (rheumatic fever) and of endopericarditis.

(*c*) By *imitation*, in schools and institutions, these spasms may spread like an epidemic.

From habit spasms, which persist for months or years in one or two groups of muscles, true *chorea* is distinguished by its involvement of the hands, feet, and other parts, by its frequent association with tonsillitis, joint pain and endocarditis (see page 485), and by its short course (eight to ten weeks on the average).

In hysterical conditions and hereditary brain defects, various other spasms occur (see below, page 499).

V. The Eyes

I shall not attempt to deal with lesions essentially local (such as a "sty"), but shall confine myself to data that have diagnostic value in relation to the rest of the body.

1. *Œdema of the Lids*

Œdema of lids, especially the lower, often accumulates in the night and is seen in the early morning, without known cause or after a debauch. In other cases it usually points to the existence of:

(*a*) *Nephritis* (prove by urinary examination).

(*b*) *Anæmia* (prove by blood examination).

(*c*) *Measles* and *whooping-cough* (eruption, paroxysms of cough).

Rarer causes are *diabetes*, *trichiniasis*, *angioneurotic œdema*, and *erysipelas.*

Trichiniasis is recognized by the presence of fever, muscular tenderness, and an excess of eosinophiles in the blood.

In angioneurotic œdema and urticaria there is usually a previous history of similar transitory swellings in other parts of the body.

The acute onset, red blush, high fever, and general prostration distinguish the œdema of erysipelas.

2. *Dark Circles under the Eyes*

may appear in any debilitated state, *e.g.*, from loss of sleep, hunger, menstruation, masturbation, etc.

3. *Conjunctivitis*

This affection forms part of hay fever, measles, streptococcus infections, typhus, trichiniasis and yellow fever. It also occurs as an independent infection. It follows overdoses of iodide of potash or arsenic. The *whole conjunctiva is reddened*, in contradistinction from the reddening about the iris seen in iritis.

Phlyctenular conjunctivitis and keratitis occurs especially in ill-nourished children of poor and ignorant parents living in congested districts, *i.e.*, under the conditions producing tuberculosis. Its cause is unknown. It produces a yellowish-red ulcerated streak growing in from the conjunctiva across the margin of the cornea.

4. *Jaundice*

Jaundice, the yellow coloration of the white of the eye by bile pigment, is easily recognized when well marked, and can be confounded only with subconjunctival fat, which differs from jaundice in that it appears in *spots and patches*, not covering the whole sclera, as jaundice does. In mild cases only the posterior portions of the sclera are tinted yellow, while the anterior part around the iris may show a bluish-white tinge in contrast. This state of things is hard to distinguish from the appearances seen in the eyes of many apparently healthy people. The presence of bile in the urine often clears up the question.

The skin, mucous membranes, urine, and sweat are also bile-stained in most cases, and the circulation of the bile in the blood often produces *slow pulse*, *itching*,[1] and *mental depression.* Lack of bile in the gut leads to flatulence and clay-colored fatty stools.

The commonest causes are: (*a*) *Biliary obstruction* (catarrh, stone or tumors obstructing the larger bile ducts, hepatic cirrhosis, or syphilis constricting them).

(*b*) *Hemolysis* (malaria, sepsis, icterus of the new-born, pernicious anæmia) producing obstruction of fine bile ducts.

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[1] In gall-stone cases one often finds itching without jaundice.

5. *Argyria*

Since the prevalent use of silver salts in the treatment of ocular disease, a brownish staining of the conjunctiva and sclera not infrequently results, even after moderate doses. Individual hypersensitiveness doubtless plays a part.

6. *The Pupils*

The *normal reflexes* to light are best tested with an electric flash light which produces a brisk and obvious contraction of the healthy pupil. To test the reaction to distance turn the patient away from the light and let him look at the farthest corner of the room. The pupil expands. Make him look at your finger a few inches distant from his eyes. The pupil contracts. Each pupil should be examined separately.

The value of the pupils in diagnosis has been greatly overestimated. There are few conditions *except tabes and paresis* in which they yield us important diagnostic evidence, for, although they are very often abnormal, the abnormalities are seldom characteristic of any single pathological condition and throw little light on the diagnosis. Especially in coma the condition of the pupils, on which much stress has been laid in textbooks, is, in my own experience, misleading or useless as a diagnostic guide.

(*a*) *The Argyll-Robertson pupil* reacts to distance, but not to light. It is of great value as a factor in the diagnosis of tabes dorsalis and dementia paralytica.

(*b*) *Dilated pupils.*—(*a*) Many phthisical patients show a more or less *transient dilatation* of one or both pupils. (*b*) *Blindness or deficient sight* (from any cause) may cause dilatation of the pupil. (*c*) Other common causes are distress or strong emotion from any cause, many fevers and comatose states, and the use of mydriatic drugs.

(*c*) *Contracted pupils* are common in old age and in photophobia from any cause. Disease high up in the spinal cord (tabes, general paralysis, etc.) may produce contraction (*spinal myosis*) by paralyzing the sympathetic dilators. *Aortic aneurism* may produce in the same way contraction of one pupil (see below, page 259).

(*d*) *Contraction with irregular outline* and sluggish reactions is often seen in iritis as a result of adhesions to the lens (posterior synechiæ), also in syphilis and without known cause.

7. *The Cornea*

(*a*) *Arcus senilis*, a grayish ring at the circumference of the cornea is one of the classical signs of old age and arteriosclerosis, but is occasionally seen in normal young adults.

(*b*) *Syphilitic keratitis*, usually seen in the hereditary form of the disease, produces an irregularly distributed haziness of the cornea, usually in both eyes and before the sixteenth year. Diagnosis depends on other evidences of syphilis.

(*c*) *Gonorrheal ophthalmia* is a common cause of opacities in the cornea and so of the types of blindness produced in infancy.

(*d*) Trauma of the eyes by cuts and blows is a not infrequent cause of scars which impair vision.

(*a*) *Ocular Motions*

(*a*) *Ptosis*, or dropping of the eyelid, is usually unilateral and dependent on paralysis of the third nerve. Its most frequent cause is basal syphilitic meningitis. The eye is usually drawn out by the action of the unparalyzed external rectus. Moderate, bilateral ptosis is common in hysterical and neurasthenic conditions, in botulism and in sleeping sickness.

(*b*) *Squint* (strabismus) is called *external* if the eye turns out, *internal* if it turns in. Of its many types and causes I mention only the acute cases due to intracranial lesions, such as tuberculous and epidemic meningitis, syphilis, tumors.

(*c*) *Nystagmus* is a rapid, usually horizontal oscillation of both eyeballs. It may be the result of albinism or of various local eye troubles, but is an important member of the symptom group characteristic of *multiple sclerosis*. It may, however, occur in other brain lesions and in health. Rarely the oscillation is vertical.

(*b*) *The Retina*

The lesions of greatest interest in general medicine are: Retinal hemorrhage, arterio-sclerosis, optic neuritis, and optic atrophy.

(*a*) *Retinal hemorrhages*, with or without other retinal changes, are important signs of *nephritis*, *grave anæmias*, and *diabetes*.

(*b*) *Arterior-sclerosis* may appear earlier or more clearly in the retina than elsewhere.

(*c*) *Papillary edema* (choked disc) is of value in the diagnosis of intracranial pressure, for example in brain tumors, tuberculous meningitis, brain abscess, and in some cases hemorrhage.

(*d*) *Optic atrophy* may be the end result of any of the types of optic neuritis just mentioned, or in a primary form is important evidence of tabes dorsalis. Many cases occur without any known cause

VI. The Nose

1. *Size and Shape.*—The enlargement of all the tissues of the nose occurring in *acromegaly* has already been mentioned. In *myxœdema* the nostrils are sometimes thickened and the whole nose loses its delicacy of shape. A *red nose* is popularly and correctly associated with *alcoholism*, but in many cases identical appearances are produced by acne rosacea or by lupus erythematosus, as well as by circulatory anomalies without any other disease.

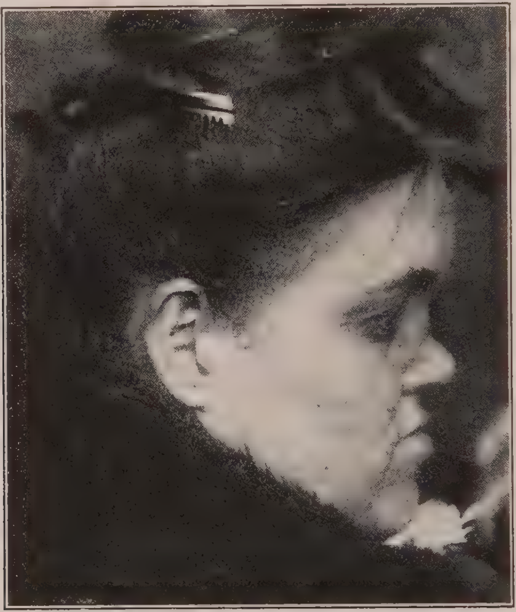

Fig. 16.—Syphilitic Depression of the Nasal Bones.

Falling in of the bridge of the nose may be due to *syphilis of the nasal bones*, especially when there are scars over the sunken portion, but is sometimes present without any disease. See Fig. 16.

Perforation of the septum (syphilitic) is often unknown to the patient and may be important in diagnosis. Occasionally it is due to mild non-syphilitic inflammations of the septum. The habit of putting a hand electric flash light against the side of the nose in the routine of physical examination is valuable.

The small, narrow nose associated with *adenoid growths* has already been mentioned.

2. The *nostrils* move visibly in many conditions involving *dyspnœa* (diseases of the heart and lungs, acute infections, etc.), and this is sometimes useful in suggesting to the physician the possibility of pneumonia, hitherto unsuspected. *Dried blood in the nostrils* may be of value as evidence of recent nosebleed.

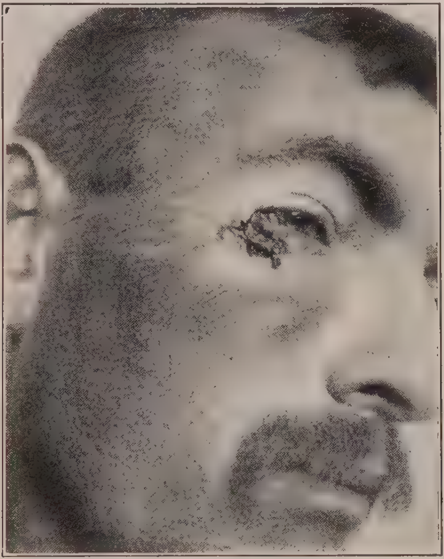

FIG. 17.—Epithelioma.

3. *Nosebleed* suggests especially trauma, vascular hypertension, infectious fevers (particularly typhoid and juvenile endocarditis); also hemorrhagic diseases (purpura, hæmophilia, acute leukæmia).

4. A *nasal discharge* in a young infant ("snuffles") suggests hereditary *syphilis*. In adults the familiar "cold in the head" may need a bacteriological examination to exclude the possibility of *nasal diphtheria* or to confirm a diagnosis of influenza.

5. A small, indolent, *long-standing sore* on the nose or near the corner of the eye should always suggest *epithelioma* (see Fig. 17) and *tuberculosis*. Microscopic examination may be necessary to determine the diagnosis.

6. The consideration of local disease within the nose does not fall within the scope of this book, but is suggested by local pain, difficulty in breathing through the nose, frequent "colds," and asthma.

(For the examination of *the ears*, see below, p. 482.)

VII. The Lips

1. *Pallor* of the mucous membrane of the lips suggests, though it never proves, anæmia. No diagnosis of anæmia should be made without at least testing the hæmoglobin (Tallqvist's scale). One minute suffices.

2. *Cyanosis*, a purplish or slatey-blue color of the lips, occurs in some healthy persons from simple "weathering." When well marked, however, it should always suggest:—(*a*) Heart disease (especially mitral or congenital lesions).—(*b*) Lung diseases (especially emphysema and pneumonia). (*c*) Polycythæmia.—(*d*) Poisoning by acetanilid or other coal-tar antipyretics, producing methæmoglobinæmia.[1]

The last is easily tested by noting the brownish (not red) tint of the blood when soaked into filter paper, as in performing Tallqvist's hæmoglobin test; the test should be confirmed by the history. Disease of the heart or lung is identified by physical examination of the chest.

3. *Parted lips*, an open mouth, may be a mere habit or may be due to nasal obstruction (adenoids). Idiots and cretins are very apt to keep their mouths open, whether there is enlargement of the tongue or not. Dyspnœa may compel a patient to keep his mouth open so as to get more air.

In cold weather a *crack* or *fissure* may appear, usually in the centre of the lower lip, and in poorly nourished individuals may persist for weeks. At the corners of the mouth fissures or cracks may be due to chapping or "*cold-sores*" (herpes), but if they persist for weeks in young children they are very suggestive of syphilis. White linear scars radiating from the corners of the mouth are presumptive evidence of healed syphilitic lesions, oftenest congenital.

4. The *mucous patches* of syphilis—white, sharply bounded areas about the size of the little-finger nail—are often seen at the junction of the skin with the labial mucous membrane, especially at the corners of the mouth.

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[1] Cyanosis of intestinal origin occurs in connection with certain diseases involving excessive intestinal decomposition. (See Gibson, Quarterly Journal of Medicine, Oct., 1907, p. 29.)

5. *Herpes* ("cold sores") is due to a lesion of the Gasserian ganglion with resulting "trophic" disturbances of the regions supplied by the trigeminal nerve. Appearing first as a cluster of vesicles ("water blisters") which break and leave a small sore near the mouth, herpes is to be distinguished by: (*a*) its distribution near the terminations of some branch or branches of the trigeminal nerve ("herpes frontalis, nasalis. labialis"); (*b*) by its lasting but a few days; and (*c*) by the absence of similar lesions elsewhere. It may be connected with a "cold" (which is often a disease of the trigeminus), with pneumonia, malaria or meningitis, but it frequently occurs without any discoverable cause. Herpetic stomatitis ("canker sores") may accompany it.

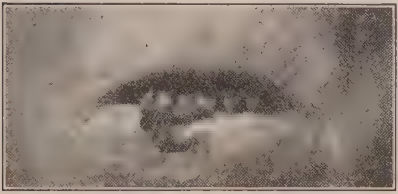

FIG. 18.—Epithelioma of the Lip.

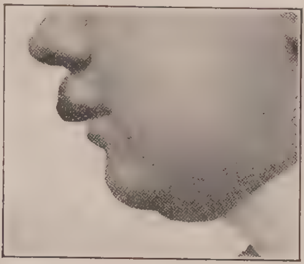

FIG. 19.—Chancre of the Lip.

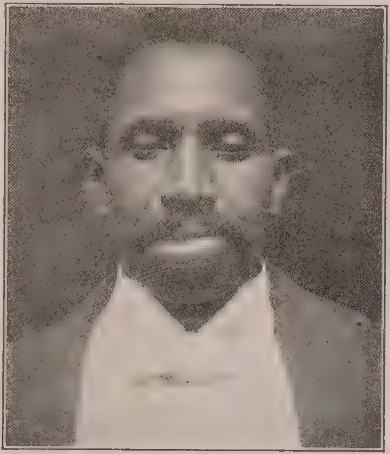

FIG. 20.—Angioneurotic Œdema of Lower Lip.

6. *Epithelioma*[1] *of the lip* and *chancre* should be suspected whenever a long-standing sore is discovered there. Epithelioma occurs almost always on the lower lip in a man past middle life (see Fig. 18). It lasts longer than chancre, is slower in producing glandular enlargement at the angle of the jaw, and is not associated with other syphilitic lesions.

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[1] It does harm to call this lesion "*cancer*" because this term is so firmly associated in the lay mind with metastasis, recurrence, and death that unnecessary suffering may result when the patient or his family learns that he has "cancer."

7. *Chancre of the lip* is commoner in women and may occur at any age, especially under forty. The sore usually lasts but a few weeks, excites early enlargement of the glands, and is usually associated with other manifestations of syphilis (see Fig. 19).

8. *Angioneurotic œdema* appears as a sudden, painless, apparently causeless swelling of the whole lip (see Fig. 20), which may attain double its normal size. The diagnosis depends on the exclusion of all known causes (trauma, infection, insect bites) and on the history of similar swellings (on the lip or elsewhere) in the past.

9. The *enlargement of the lips* in myxœdema and cretinism has been mentioned above (page 19).

10. *Hare-lip* is a vertical slit (congenital deficiency) in the upper lip opposite to the nostril; it is often connected with an antero-posterior cleft through the hard palate ("cleft palate"). The lesion may be double, leaving a small island of tissue continuous with the nasal septum (intermaxillary bone). Diagnosis is made at a glance.

VIII. The Teeth

The first set of teeth is fairly constant in its order and date of appearance. In Fig. 21 the number of the month when each tooth is most apt to appear is marked on the tooth. The second set (permanent teeth) arrives (less regularly) between the sixth and the fifteenth year, except the "wisdom teeth," which appear about the twenty-first year.

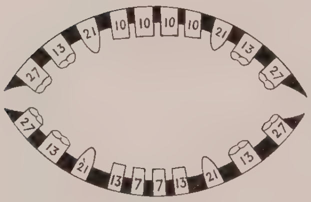

Fig. 21.—Diagram Showing the Month at which Each Tooth (of the First Set) Should Appear.

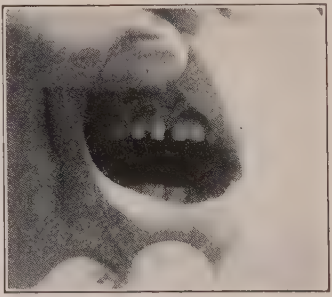

Fig. 22.—Notched Incisors in Congenital Syphilis.

1. *Rickets* or cretinism often delays dentition considerably.

2. *Congenital syphilis* may be associated with various deformities of the central incisors (permanent). The most constant deformity is that shown in Figs. 22 and 22a.

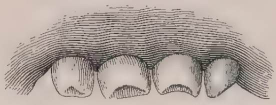

FIG. 22a.—Hutchinsonian Teeth.

3. *Teeth-grinding.*—Nervous, delicate, oversensitive children often grind their teeth in their sleep. There is no foundation for the popular superstition that this act indicates "worms."

IX. The Breath

Foul breath is oftenest due to local causes such as:

(*a*) Foul teeth and gums (neglect, Riggs' Disease).
(*b*) Stomatitis of any variety.
(*c*) Follicular Tonsillitis with cheesy deposits in the crypts.
(*d*) Nasal Disease.

Rarer causes are *abscess or gangrene of the lung*, in which the breath may be intensely foul; the source of the odor is made evident by the sputa.

Acetone breath has a faintly sweetish odor, which has been compared to that of chloroform, new-mown hay, and rotting apples. It occurs not only in diabetes, but in various conditions involving starvation (vomiting, fevers), and especially, but not only, a lack of carbohydrates.[1]

In *uræmia* a foul odor is often noticed, and an ammoniacal ("urinous") smell has been mentioned by many writers. In *typhoid* and in *syphilis* some persons seem to detect a characteristic odor, but the evidence is insufficient. *Alcoholic breath* is often of value in correcting the false statements of its possessor. In comatose persons we must remember that a drink may have been taken just before an attack of apoplexy or any other cause for coma, so that an alcoholic breath in comatose patients does not prove that the coma is due to alcohol.

In *poisoning by illuminating gas* the gaseous odor of the breath may be noticed.

X. The Tongue

The *act of protruding the tongue* may give us valuable information on the condition of the nervous system.

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[1] See Taylor: "Studies on an Ash-free Diet." University of California Publication, July 30th, 1904.

(*a*) The *hesitating, tremulous* tongue of typhoidal states is very characteristic. Simple tremor is seen in alcoholism, dementia paralytica, and weakness. A *tongue protruded very far* means usually a neurasthenic individual who is in the habit of examining it in a looking glass.

(*b*) If the *tongue is protruded to one side*, it usually means facial paralysis as part of a hemiplegia; rarely it is due to lesions of the hypoglossal nerve or its nucleus (in bulbar paralysis or tabes).

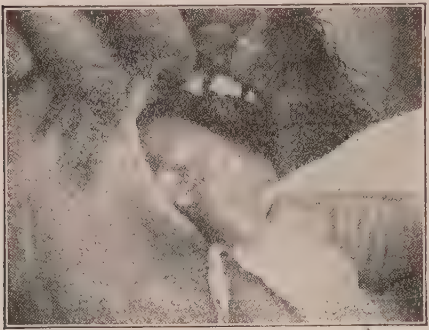

FIG. 23.—Syphilis of the Tongue.

(*c*) A *coated tongue* (due mostly to lack of saliva) is not often of much value in diagnosis, and there is no need to distinguish the varieties and colors of coats; but a few suggestions may be obtained from it. Many persons who seem otherwise perfectly healthy have coated tongues in the early morning. This is especially true in mouth-breathers, in smokers, and in those who keep late hours.

In those whose tongues are usually clean the appearance of a coat is associated often with arrested digestion, constipation, or fevers.

A *clean tongue in a dyspeptic* suggests peptic ulcer. This point I have found of more value than any inference from a coated tongue.

A *dry, brown-coated*, perhaps cracked tongue goes with uræmia, exhausted states and wasting diseases with or without fever.

(*d*) *Cyanosis* and *jaundice* may be seen in the tongue but better elsewhere.

(*e*) *Indentation* of the edges of the tongue by the teeth occurs especially in foul, neglected mouths, but has no diagnostic value.

(*f*) *Herpes* ("canker") often occurs on the tongue; it begins as a group of vesicles, but these rupture so soon that we usually see first a very small, grayish ulcer with a red areola. It heals in a day or two, *i.e.*, more quickly than the syphilitic mucous patch or any other lesion with which it is likely to be confounded.

(*g*) *Cancer*, *tuberculosis*, and *syphilis* may attack the tongue and form deep, long-standing ulcerations. *Syphilis* can usually be diagnosed by the history, the presence of other syphilitic lesions, the Wassermann reaction, and the therapeutic test (see Fig. 23). *Cancer* and *tuberculosis* should be diagnosed by microscopic examination, though cancer is more commonly found in men (especially smokers) past middle life and on the side of the tongue. A local reaction after the injection of tuberculin may be of decisive importance.

(*h*) "*Simple ulcers*" are due to irritation from a tooth or to trauma, and heal readily if their cause is removed.

(*i*) *Deep fissures* of the tongue are sometimes due to syphilis, which is recognized in other lesions, by history, or by the Wassermann test.

(*j*) *Leukoplakia buccalis* (lingual corns) refers to whitish, smooth, hard patches of thickened epithelium, usually on the dorsum of the tongue is smokers, running a chronic course without pain or ulceration, but important because epithelioma has been known (and not rarely) to develop in them.

(*k*) *Geographic tongue* is a desquamation of the lingual epithelium in sinuous or circinate areas, which spread and fuse at their edges, while the central portions heal, giving a look something like the mountain ranges in a geographical map. It usually gives no trouble unless the patient's attention becomes concentrated on it.

(*l*) *Hypertrophy* of the tongue has already been mentioned in connection with myxœdema and cretinism. It may occur independently as a congenital affection.

XI. THE GUMS

(*a*) A *lead line* should be looked for in every patient as a matter of routine, as it may not be suggested by anything in the patient's symptoms or history, yet may be the key to the whole case.

The deposit of lead sulphide in (not on) the gums is not blue, but gray or black; and is not a line, but a series of dots and lines arranged near the free margin of the gums. Where there are no teeth there is no lead line. In faint or doubtful cases a hand lens is of great assistance and shows up the dotted arrangement of the deposit very clearly (see Fig. 24). It is unfortunate that the term "blue line" has become attached to these gray-black dots.

(*b*) A *bismuth line*—as in poisoning from the injection of bismuth paste—may present all the appearances of a lead line, though in some cases the staining is more diffuse and occurs at some distance from a tooth as well as at the free margin of the gum. The analyses of the feces and the history of the case serve to distinguish it from a lead line.

(*c*) *Sordes*, a collection of epithelium, bacteria, and food particles, accumulates about the roots of the teeth with great rapidity in febrile cases, but has no considerable diagnostic importance.

(*d*) *Spongy and bleeding gums* occur as part of the disease "*scurvy,*" after overdoses of mercury or potassic iodide, in various debilitated states, and sometimes without known cause. The teeth are loosened and the flow of saliva is usually profuse. The stench from such cases is often intolerable.

(*e*) *Suppuration* about the roots of the teeth (*pyorrhœa alveolaris*) is common in elderly people and in neglected mouths, and seems in some cases to have some constitutional effects; but in most cases its effects appear to be wholly local.

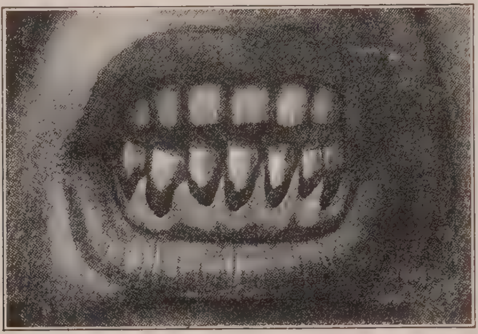

FIG. 24.—Lead-dots in the Gums. (Use hand lens in looking at this picture and examine the gums not the teeth.)

(*f*) *Gumboil* (*alveolar abscess*), is easily recognized by the familiar signs of abscess associated with a diseased tooth. Dental abscesses are an important cause of obscure fevers and joint pains.

(*g*) "*Epulis*" is a word applied to various soft tumors springing from the jaw bone or occasionally from the gums themselves. Many of them are sarcomatous, but microscopic examination is necessary to distinguish these from fibroma, granuloma, and angioma.

XII. THE BUCCAL CAVITY

1. *Eruptions*

(*a*) *Koplik's spots* in *measles* are of much importance. They appear chiefly in the inside of the cheeks, opposite the line of closure of the molars, and consist of minute, bluish-white spots, each surrounded by a red areola and sometimes fusing into larger red areas.

(*b*) The *syphilitic mucous patch* (see above) should be looked for in suspicious cases, not only in easily accessible parts of the mouth, but round the roots of the gums, where the cheeks or lips have to be pushed away to afford a good view.

2. *Pigmentations*

In Addison's disease brown spots or patches often occur on any part of the mucous membrane of the mouth. They may also occur in negroes without any disease and after ulcerations (*e.g.*, from a tooth), so that they are not distinctive of Addison's disease.

3. *Gangrene*

Gangrene (stomatitis gangrenosa, "noma"), a rare disease of weakly children, starts as a hard red spot inside the cheek and usually not far from the corner of the mouth (see Fig. 25). There is a swelling of the whole cheek, especially under the eye. The odor of gangrene is usually the first thing to make clear the diagnosis. Then the gangrene appears externally as a black patch on the cheek, surrounded by a red halo.

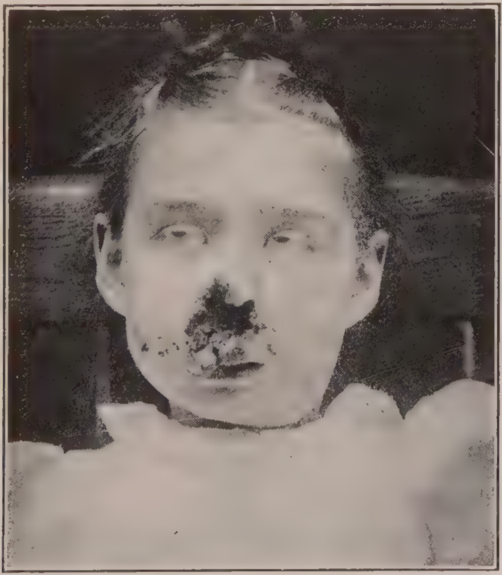

FIG. 25.—Gangrenous Stomatitis ("noma").

XIII. THE TONSILS AND PHARYNX

METHOD OF EXAMINATION.—Place the patient facing a good light, natural or artificial. An electric flash light is especially convenient. Ask him to open his mouth *without protruding the tongue.* Ask him to say "Ah." Then gently press down and forward on the dorsum of the tongue (not too far back) with a spoon or tongue depressor,[1] until a good view of the throat is obtained.

Look especially for:

1. Inflammations (redness, eruptions, spots, or membranes).
2. Ulcerations.
3. Swellings.
4. Reflexes.

1. *Inflammations*

(*a*) *General redness* means a mild or early pharyngitis, but may precede severe diseases like diphtheria and scarlet fever.

(*b*) *Yellowish-white spots* on the tonsils, more or less confluent, mean *follicular tonsillitis* in the vast majority of cases, but only by culture can we exclude diphtheria with certainty. Fever and headache are usually present.

(*c*) A *membrane*, continuous and grayish-white over one or both tonsils, especially if it extends to soft palate and uvula, means diphtheria in almost every case.[2] Rarely a similar membrane is seen in streptococcus throats with or without scarlet fever. Cultures alone can decide.

(*d*) The *eruptions* of smallpox and chickenpox may be distributed in the pharynx as well as over the rest of the respiratory tract. They are recognized by association with more characteristic skin lesions and constitutional signs.

2. *Ulcerations*

(*a*) Deep chronic, painless or but slightly painful ulcerations of the tonsils or soft palate are oftenest due to *syphilis.* Improvement under treatment and the manifestations of syphilis elsewhere make the diagnosis possible.

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[1] If the patient is especially nervous, it is sometimes well to let him press down his tongue with his own forefinger.

[2] *Thrush*, a rather rare disease of ill-nourished infants, due to a fungus of the yeast order, may produce on the pharynx, tongue, or in any part of the mouth, patches of white membrane. As the disease is almost wholly local and without constitutional manifestations, it is passed over briefly here.

Streaks of *mucus* or bits of *milk coagulum* are sometimes mistaken for a membrane.

(*b*) *Tuberculosis* may produce similar deep ulcerations, easily recognized by their association with tuberculosis of the lung or larynx. Occasionally smaller "miliary" tubercles, not unlike "canker sores," are seen in the tonsillar region. Tuberculous lesions are usually very painful and tender, syphilitic lesions almost free from tenderness. The chronic course of pharyngeal tuberculosis and the presence of other tuberculous lesions identify it.

(*c*) *Malignant disease* (oftenest sarcoma) may attack the tonsil, and forms a painful, tender and finally ulcerating tumor. No other lesion of the tonsil grows so fast and invades surrounding parts so extensively except abscess; in abscess the pain, fever, and constitutional manifestations are far greater.

3. *Swellings*

(*a*) *Chronic swollen or protruding tonsil* (unilateral or bilateral) without fever or constitutional symptoms may represent hypertrophy following acute tonsillitis or may be part of the general adenoid hypertrophy common in children's throats. Tonsils may be small or buried and yet full of pus. Their size is of no particular importance in judging of the harm done by them.

(*b*) *Acute swollen tonsil* is usually part of follicular tonsillitis (see above), but may occur without spots, and often accompanies scarlet fever. Swelling, pain in swallowing, and fever are the essentials of diagnosis. Our chief care should be to exclude:

(*c*) *Peritonsillar abscess* (quinsy sore throat). Here the swelling is usually unilateral and greater than in follicular tonsillitis. The *pain*, which is often severe, is continuous and not merely on swallowing. Fever, constitutional symptoms, and swelling of the glands at the angle of the jaw are all more marked than in follicular tonsillitis. The *pain*, which is often severe, is continuous and not merely on swallowing. Fever, constitutional symptoms, and swelling of the glands at the angle of the jaw are all more marked than in follicular tonsillitis. The voice is nasal or suppressed, and there is often salivation. The pillars of the fauces and the soft palate take part in the swelling and the throat may be almost blocked by it. The suffering increases until the abscess breaks or is opened. Fluctuation is often late and indefinite, but should always be sought for.

(*d*) *Retropharyngeal Abscess.*—A swelling in the back of the pharynx near the vertebra occurs not infrequently during the first year of life. A peculiar cry or cough, like the bark of a puppy or the call of a heron, is very often associated (the French "cri de canard"). The parents are often unaware that the throat is the seat of the trouble, and only digital examination proves the presence of bulging and fluctuation, usually on one side of the posterior pharyngeal wall.

A similar abscess of chronic course may complicate tuberculous cervical caries (see below, page 31).

(*e*) *Swollen uvula*, with transparent œdema of its tip, often complicates a pharyngitis or any lesion with violent cough. *Elongation of the uvula* may bring it into contact with the tongue and by tickling excite cough.

(*f*) *Perforation of the soft palate* or its *adhesion to the back of the pharynx* means *syphilis* almost invariably, and, as it may be the only sign of an old infection, it is a valuable piece of evidence.

4. *Reflexes*

(*a*) *Lively or exaggerated pharyngeal reflexes*, such that the patient gags and coughs as soon as one touches the dorsum of the tongue, are seen in many normal persons and in many alcoholics. It is this condition, combined with a smoker's pharyngitis, that leads to many cases of morning vomiting in alcoholics.

(*b*) *Diminished or absent reflexes* (with paralysis of the palate) occur in postdiphtheritic neuritis and bulbar paralysis. Fluids are regurgitated through the nose and the voice has a peculiar intonation.

To test for paralysis, ask the patient to say "Ah." In unilateral paralysis one side of the palate remains motionless; in bilateral paralysis the whole palate is still.

(*c*) Diminished or absent gag-reflex is often seen (without palatal paralysis) in "nervous" people and neurasthenic states, associated with corneal anæsthesia and lively knee jerks.

XIV. THE NECK

Long, thin necks are often seen in phthisical individuals, and short necks in the barrel-chested, but nothing more than a bare hint can be derived from such facts. The lesions oftenest searched for in the neck are: 1. *Enlarged glands* (cervical adenitis). 2. *Abscesses and scars*. 3. *Thyroid tumors*. 4. *Pulsations* (see below page 89). 5. *Torticollis* and other lesions simulating it. 6. *Tuberculosis* of the cervical vertebræ. 7. Evidence of *meningitis*.

Rarer lesions will be mentioned below.

1. *Chains of Enlarged Glands*

radiate in all directions from the angle of the jaw—upward, in front of the ear and behind it, forward along the ramus of the jaw, and downward to the clavicle. The areas drained by the different groups overlap so much that it is not necessary to distinguish them.

The commonest causes of enlargement are:

(*a*) *Tonsillitis and other inflammations within or around the mouth* (diphtheria, the exanthemata, "cankers," carious teeth, etc.). Glandular swellings due to these causes are usually acute and more or less tender; most of them disappear in a fortnight or less, but some persist (without pain) indefinitely.

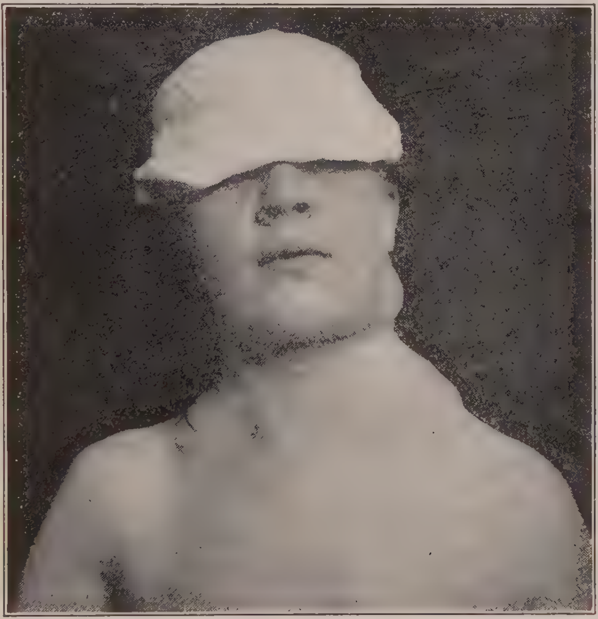


FIG. 26.—Tuberculous Glands.

(*b*) *Tuberculosis;* long-standing cervical adenitis in children and young adults, with a tendency to involve the skin and to suppurate, is usually tuberculous. Certain diagnosis needs microscopic examination, animal inoculation, the tuberculin test and the Wassermann.

(*c*) *Syphilis;* small, non-suppurating glands, occurring in the neck and about the occiput in adults, often accompany syphilis, but the diagnosis depends on the presence of unmistakable syphilitic lesions elsewhere. Occasionally syphilitic glands are large and soft.

(*d*) *Hodgkin's disease;* chronic, large, rarely suppurating glands in the neck, axillæ, and groins, with slight splenic enlargement and normal blood, suggest Hodgkin's disease, but microscopic examination is necessary to exclude tuberculosis. A superficial gland can be excised under cocaine, with very little pain.

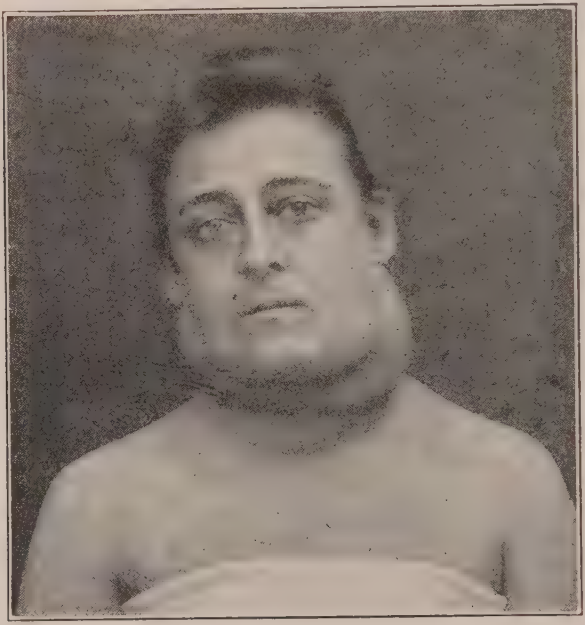


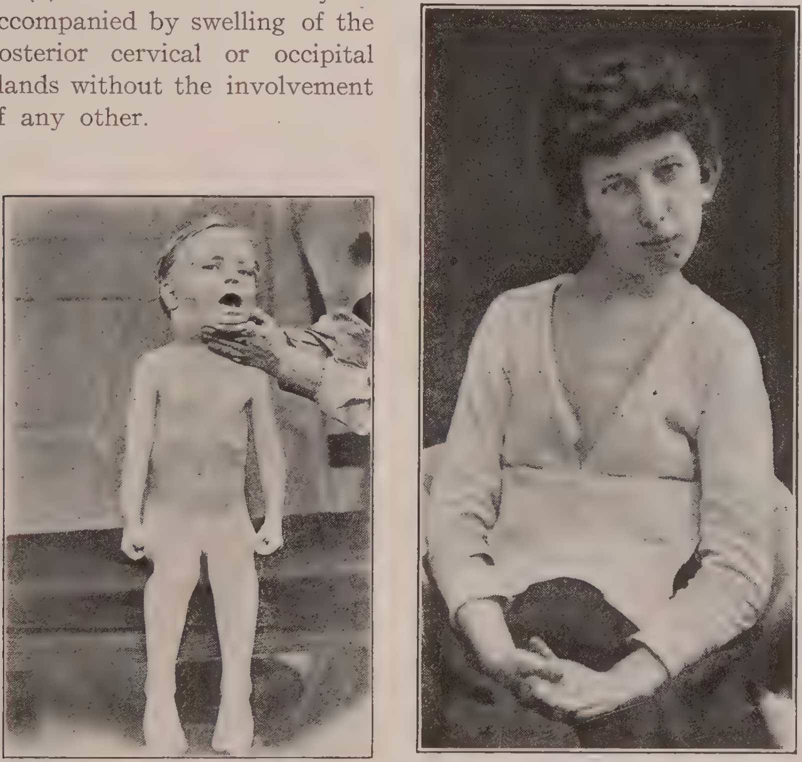
FIG. 27.—Hodgkin's Disease, Six Months Duration.

(*e*) *Lymphoid Leukæmia.* No distinguishing characteristics can be found in the glands, but any nodular enlargement in the neck should lead us to examine a film specimen of blood; the leukæmic blood changes can be easily and quickly recognized.

(*f*) *Malignant disease* (near by or at a distance) may enlarge the cervical glands. Cancer of the lip or tongue, sarcoma of the tonsil, and, among distant lesions, cancer of the stomach or pleura have caused enlargement of these glands in cases under my observation.

(*g*) If the *parotid gland* alone is swollen and there are fever and pain on chewing, the case is probably one of *mumps*, especially if there are other cases in the vicinity. Malignant disease and ordinary sepsis may also attack the parotid.

(*h*) *German measles* may be accompanied by swelling of the posterior cervical or occipital glands without the involvement of any other.



FIGS. 28 and 29.—Cervical Abscess in Pott's Disease. (Bradford and Lovett.)

2. *Abscess or Scars*

Abscess or scars in the sides and front of the neck generally result from glandular tuberculosis; hence the presence of scars may be of value in the diagnosis of doubtful cases with a suspicion of tuberculosis in later life. Aside from glandular abscesses (tuberculous or septic) it is rare to find any suppuration in the neck, except in the nape, where deep, septic abscess (carbuncle) and superficial boils are common. High Pott's disease may be complicated by abscess (see Figs. 28 and 29)

3. *Thyroid Tumors*

occur chiefly in two diseases;

(*a*) *Simple goitre* (unilateral or bilateral).

(*b*) *Goitre with exophthalmos, tachycardia*, and tremor, sweating and loss of weight (Thyrotoxicosis or Graves' disease).

The tumor may look the same in these two diseases (see Fig. 30); it varies in outline and consistency according to the amount of gland tissue and fibrous or cystic degeneration that is present. Owing to its connection with the larynx it moves up and down somewhat when the patient swallows, but is not attached to any other structures in the neck. The enlargement is often unilateral or largely so. If very vascular, the tumor may vary greatly in size from moment to moment or at certain times (*i.e.*, menstruation, pregnancy).

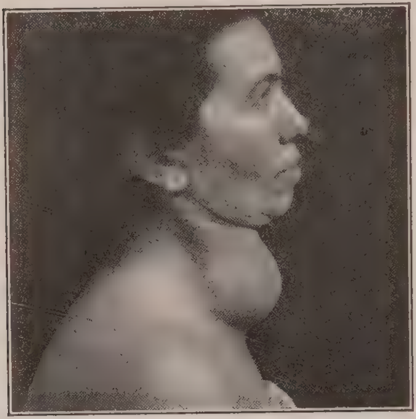


FIG. 30.—Simple Goitre.

Since the normal thyroid can rarely be felt, *atrophy of the gland* (as in myxœdema) is unrecognizable.

Cancer or sarcoma also occur in the thyroid and may be difficult to distinguish from goitre. Malignant tumors are usually painful, grow fast, are accompanied by emaciation and anæmia, are often harder and more nodulated than benign goitres, and invade the neighboring tissues and lymphatics. Histological examination should decide in doubtful cases.

4. *Torticollis* (*Wry-neck*) *and Other Lesions Resembling It*

(*a*) *Spasm* (tonic, rarely clonic) of the sterno-mastoid and trapezius may be due to irritation of the spinal accessory nerve by swollen glands, abscess, scar, or tumor, but more often occurs without known cause ("rheumatic" and nervous" cases). The muscle is rigid and tender.

(*b*) *Congenital torticollis* (a counterpart of club-foot) is due to *shortness* of the muscle *without spasm*. It is almost always right-sided and associated with facial asymmetry.

(*c*) *Dislocation of the upper cervical vertebræ* causes a distortion of the neck much like that of torticollis (see Fig. 31). The diagnosis depends on the history of injury, the absence of true muscular spasm and the *x*-ray picture.

(*d*) *Compensatory cervical deviations:* (1) When there is marked *lateral curvature* of the spine, *with or without Pott's disease*, the head may be inclined so far to the opposite side that torticollis is simulated, (see below, page 72). (2) When the power of the two eyes is markedly different, as in some varieties of astigmatism, the head may be habitually canted to one side to assist vision. (3) In some cases due to none of the above causes, habit or occupation (heavy loads on one shoulder) seem to produce the condition.

(*e*) *Forced attitude* from *cerebellar disease* may resemble torticollis. The diagnosis depends on the other evidences of intracranial disease.

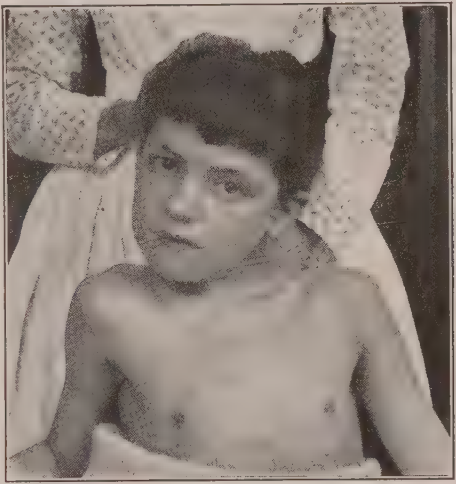


FIG. 31.—Dislocation of the Cervical Vertebræ. (Walton.)

5. *Cervical Pott's Disease (Vertebral Tuberculosis)*

has the characteristics alluded to below in the section on joint tuberculosis, viz., stiffness due to muscular spasm, malposition of the bones and of the head, and abscess formation (see page 33).

Diagnosis depends on wry-neck with stiffness of the muscles of the back and neck and pain in the occiput—a very characteristic symptom-group. The chin is often supported by the hand. "Rheumatic" or traumatic torticollis, however, may present all these symptoms, and diagnosis may be impossible without the aid of time and therapeutic tests.

6. *Branchial Cysts and Fistulæ*

These, due to persistence of parts of the fœtal branchial clefts, are not very uncommon (see Fig. 32).

A branchial cyst is a globular or ovoid fluctuating sac, hanging or projecting from the side of the neck or the region of the hyoid bone, painless and slow of growth. It may transmit the motions of the carotids and be mistaken for aneurism, but has no expansile pulsation and occurs in youth, when aneurism is practically unknown. Some such cysts may be emptied by external pressure.[1]

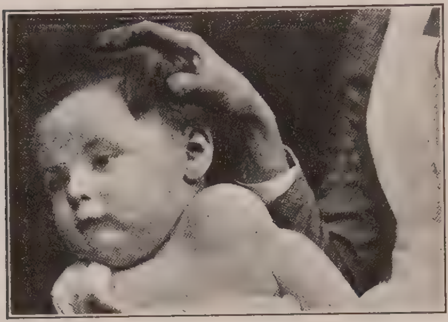


FIG. 32.—Branchial Cyst.

Branchial cysts may contain serous, mucous, or serosanguineous fluid, or hair and sebaceous material, according as their lining wall is derived from ectoderm or entoderm. Diagnosis depends on the position and consistency of the growth and on the results of aspiration.

Branchial fistulæ (congenital) may open externally in the neck, and occasionally are complete from neck to pharynx. They may become occluded and suppuration result.

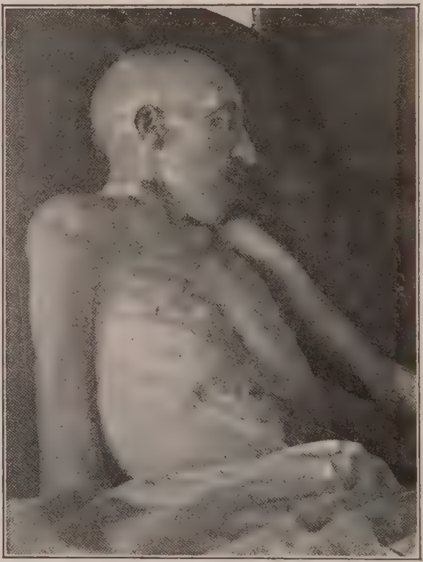


FIG. 33.—Mediastinal Neoplasm with Cervical Metastases and Obstructed Vena Cava.

7. *Actinomycosis*

Actinomycosis, though it usually arises in the lower jaw bone, may appear externally in the neck. A denser infiltration with bluish-colored, semifluctuating areas in it, but without any distinct lumps or sharp outlines, is strongly suggestive of actinomycosis, and should always lead to a microscopic examination of excised portions or of the discharge.

Fistulæ may form, but are less common than in tuberculosis.

8. *A Cervical Rib*

springing from the seventh cervical vertebra and ending free or attached to the first thoracic rib, appears in the neck as an *angular fulness which pulsates*, owing to the presence of the subclavian artery on top of it. It rarely produces any symptoms and is generally encountered when percussing the apex of the lung. The bone can be felt behind the artery by careful palpation and demonstrated by radiography. Pain or wasting in the arm, and occasionally thrombosis or gangrene may occur and may be cured by removing the rib.

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[1] A patient of mine can produce a gush of foul fluid in the mouth by pressure over a small cyst in the neck.

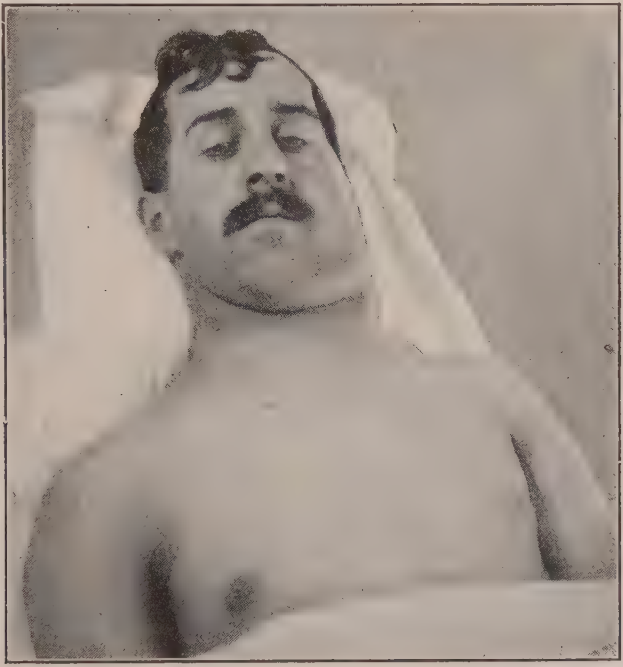

FIG. 33a.—Anthrax Infection of the Neck.

9. *Inflammatory or Dropsical Swelling of Neck*

Venous thrombosis, aneurism, mediastinal tumors and inflammatory exudates (see Fig. 33a) may produce œdema in the neck.

CHAPTER III

THE ARMS AND HANDS; THE BACK

THE ARMS

Most of the lesions of these parts are *joint lesions* and are dealt with in the section on *joints*. Others fall under the province of the neurologist or the dermatologist, but must be briefly mentioned here.

I. Paralysis of One Arm

Paralysis of most or all the muscles *of one arm* occurs oftenest in: (*a*) *Hemiplegia*—with paralysis of the leg and often of the face on the same side. (*b*) *Pressure neuritis*—traumatic or from new growths. (*c*) *Obstetrical paralysis*—neuritis from injury during parturition. (*d*) *Lead or alcoholic neuritis*—extensors of wrist especially, and often in both arms. (*e*) *Anterior poliomyelitis*—infantile paralysis. (*f*) *Hysteria and traumatic neuroses.*[1] (*g*) *Subdeltoid bursitis* is a common result of falls in which one strikes upon the shoulder, and of other injuries to the upper arm, though it may come on without any known cause. A soreness and stiffness of the shoulder is soon followed by inability to use the deltoid or to lift the arm from the side. Atrophy supervenes with astonishing rapidity and recovery may be a matter of many weeks. Disuse is undoubtedly a factor in this.

Pressure Neuritis.—The history of the case is of the greatest importance. During surgical anæsthesia the brachial plexus or the musculo-spiral nerve may be compressed, and paralysis is noted as soon as the patient comes out of anæsthesia. In a similar way in deep sleep, especially *drunken* sleep with the arm hanging over a bench or doubled under the body, the nerves may be injured. Pressure from a *crutch* or from the head of the humerus in *fractures* or *dislocations*, or even a *violent fall on the shoulder* without injury of bones, may result in a paralyzed arm (with or without subdeltoid bursitis).

Diagnosis rests on the history, and on the fact that not only the muscles of the shoulder group and the extensors of the wrist are affected, but also the *supinator longus*, while in the toxic paralyses, especially lead, the supinator longus is spared.

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[1] Less common are paralyses due to lesions of the arm centre in the cerebral cortex (tumor, softening, cyst abscess, hemorrhage, thromboses, or embolism).

Obstetrical Neuritis.—In instrumental deliveries or when forcible traction on the child's arm has been necessary, a paralysis of the arm often results, with or without fractures, and, what is important, is often not noticed till some years later, and then thought to have just arisen; thus it may be mistaken for anterior poliomyelitis or other lesions.

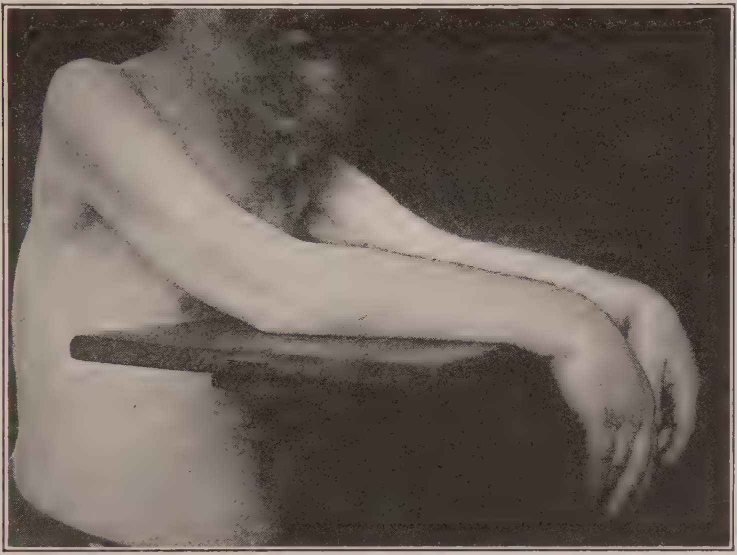

FIG. 34.—Wrist Drop, following Lead Neuritis.

Toxic Neuritis.—Lead or alcohol produces usually a weakness of *both* forearms, especially the extensors of the wrist ("wrist-drop"), but one side may be predominantly affected and other muscles are involved in most severe cases. The history, the other signs of lead poisoning, and the soundness of the supinator longus distinguish it from other paralyses. (See Fig. 34.)

All these forms of neuritis are apt to be accompanied either by pain, by anæsthesia, or by paræsthesia, which helps to distinguish them from the cerebral and spinal paralyses next described.

Acute Anterior Poliomyelitis.—Paralysis attacks a child suddenly and without apparent cause, usually after "a feverish turn." Either the upper arm group (deltoid, biceps, brachialis anticus, and supinator longus) or the lower arm group (flexors and extensors of wrist and fingers) may be affected. The arm is flabby and painless, the muscles waste rapidly, and the electrical reactions show degeneration, often within a week.

Hysterical and Traumatic Neuroses.—The history and mode of onset, the frequent association of sensory symptoms which do not fit the distribution of any peripheral nerve, spinal segment, or cortical area, the normal reflexes and electrical reactions distinguish most cases of this type, but diagnosis is sometimes impossible.

Paralysis of both arms is much less common than paralysis of one arm, and occurs chiefly in poisoning by lead and in multiple neuritis. Rarely it is seen in the late stages of chronic diseases of the spinal cord.

II. Wasting of One Arm

(*a*) *Rapid atrophy* occurs in all the types of neuritis mentioned, also in subdeltoid bursitis, poliomyelitis and progressive muscular atrophy. In the latter it occurs without complete paralysis, though the wasted muscles are weak. Progressive muscular atrophy usually begins in the muscles at the base of the thumb and between it and the index finger. Less often the disease begins in the deltoid. In either case the rest of the arm muscles are later involved.

In the atrophies just mentioned a lack of *trophic* or nourishing functions which should flow down the nerve is usually assumed to explain the wasting ("*trophic atrophy*"). From this we distinguish the atrophy due simply to *disuse* of the muscles without nerve lesions.

(*b*) *Slow atrophy of disuse* occurs in the arm in *hemiplegia*, infantile or adult, and in other cerebral lesions involving the arm centre or the fibres leading down from it.

(*c*) *Cervical rib* occasionally leads to wasting as well as pain in the corresponding arm.

(*d*) The atrophy often seen in *hysterical* cases is probably due to disuse and is similar to that occurring in an arm that has been splinted after fracture or dislocation.

III. Contractures of the Arm

After cerebral lesions involving the arm centre, and in almost any spinal or peripheral nerve lesion which involves one set of muscles and spares another, the sound muscles contract (or overact) and permanent deformities result. In hysteria similar contractures occur. Contractures have in themselves little or no diagnostic value, but indicate a *late and stubborn stage* of whatever lesion is present.

IV. ŒDEMA OF THE ARM[1]

Causes.—1. Clot of axillary or brachial vein, usually from heart disease or from: 2. Pressure of scar tissue or of tumors—aneurism, cancer of axillary glands, lung or mediastinum, Hodgkin's disease. 3. Nephritis, when the patient has lain long on one side. 4. Inflammation, usually with evidence of lymphangitis spreading up the arm from a septic wound on the hand. 5. Deep axillary abscess—an insidious painful septic focus, not depending on tuberculosis or on any form of adenitis, may burrow so deeply in the axilla that œdema of the arm (as well as pain) is produced. Leucocytosis and slight fever accompany it. The diagnosis is easily made from the above data provided we are aware of the existence of this uncommon but distinct clinical entity.

The diagnosis of the *cause of œdema* is usually easy in the light of the facts brought out by the general physical examination (heart, urine, local lesions, etc.).

The *arteries of the arm* (brachial and radial) are to be investigated for changes in the vessels (see page 90) and for the evidence given by their pulsations as to the work of the heart (see page 102).

V. TUMORS OF THE UPPER ARM

In the upper arm we have: 1. Fatty tumors. 2. Sarcoma of the humerus. 3. Ruptured biceps. 4. Syphilitic nodes on the humerus. 5. Tuberculosis of the humerus. 6. Gouty deposits in the triceps tendon.

Fatty tumors are recognized by the history of long duration and very slow growth, by their superficial position, usually external to the muscles, and soft, lobulated feel.

Sarcoma forms the only large tumors springing from the humerus. It is usually hard and obviously deep seated (see Fig. 35).

Ruptured biceps. The lower half of the biceps projects sharply when the muscle is contracted, looking as if the biceps had slid down from its normal site. This appearance suddenly following a wrench or strain of the biceps is diagnostic.

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[1] Distinguished, like all œdema, by the fact that a dent made by pressing with the finger does not at once disappear when the pressure is removed.

Syphilitic nodes are flattened elevations on the bone, usually about the size of a half-dollar, and feel like the callus after a fracture, but project only from one side of the bone. There is pain, especially at night, and moderate tenderness. A history or other and more characteristic lesion of syphilis, a radiogram or a Wassermann reaction may be necessary for diagnosis.

Tuberculous lesions[1] are much more common on the forearm bones, but are occasionally seen on the humerus near the epiphyseal ends. They usually involve and perforate the skin, leaving an indolent, suppurating sinus leading to necrosed bone. The evidence of tuberculosis in other organs and the slow, "cold" progress of the lesion assist the diagnosis. In doubtful cases the local reaction after the subcutaneous injection of tuberculin may be of distinct value. Pain, tenderness,

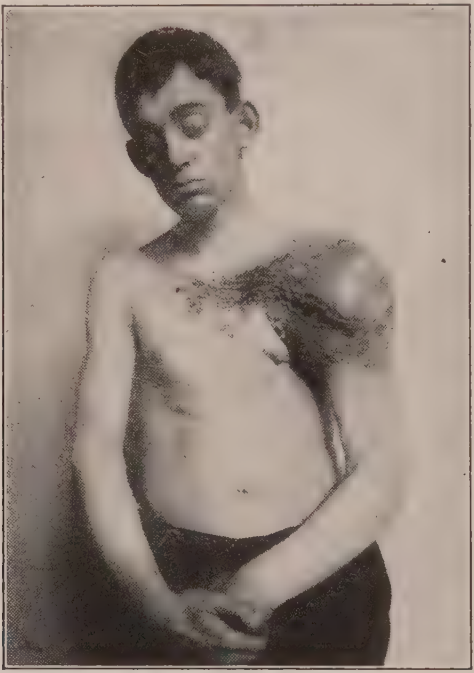

Fig. 35.—Sarcoma of Humerus.

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[1] *Coccidioidal Granuloma,* a rare disease clinically identical with tuberculosis, but due to a wholly different organism, an animal parasite resembling a coccidium, has been described by Rixford, Gilchrist, Montgomery, and others.

œdema, redness and heat may then appear or may be increased if already present.

Gouty tophi are sometimes seen along the fasciæ covering the triceps tendon. They are hard and painless. The diagnosis depends upon the peculiar situation of the lesions and their association with other evidences of gout.[1]

VI. Miscellaneous Lesions of the Forearm

Bowing of the forearm bones occurs in rickets and in Paget's disease (see Fig. 242, p. 450). The lesions in the other parts of the body make the diagnosis clear.

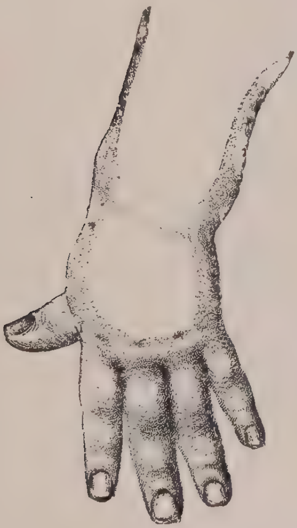

Fig. 36.—Rachitic Epiphysitis.

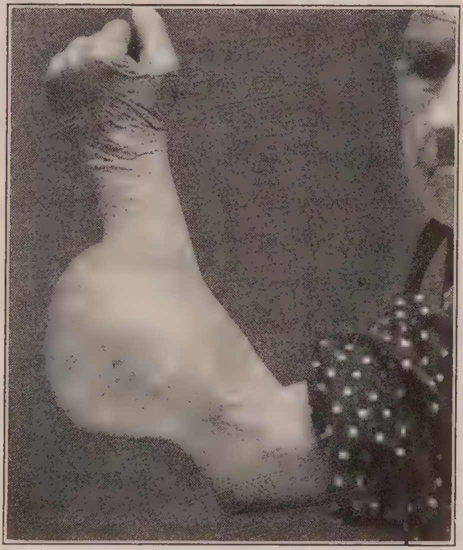

Fig. 37.—Sarcoma of Ulna.

Local lesions of the bones of the forearm are chiefly tuberculous and syphilitic, both of which have been sufficiently described in the last section.

In the wrist bones we find:

1. *Rachitic enlargement of the epiphyses.* In rickets the terminal epiphyses at the wrists take part in the general epiphyseal enlargement

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[1] Bursitis over the olecranon ("miner's elbow") produces a tender fluctuating swelling over the tip of the elbow.

so common in the disease. The diagnosis is easy, for there is no other disease of infancy producing general enlargement of the epiphyses (see Fig. 36).

2. *Hypertrophic pulmonary osteoarthropathy* (Figs. 38, 39, and 40). An enlargement of the lower ends of the radius and ulna, with clubbing of the fingers (see below, page 54), is recognized by the x-ray picture and by its association with pulmonary or pleural diseases of many years' duration (bronchiectasis, phthisis, empyema).

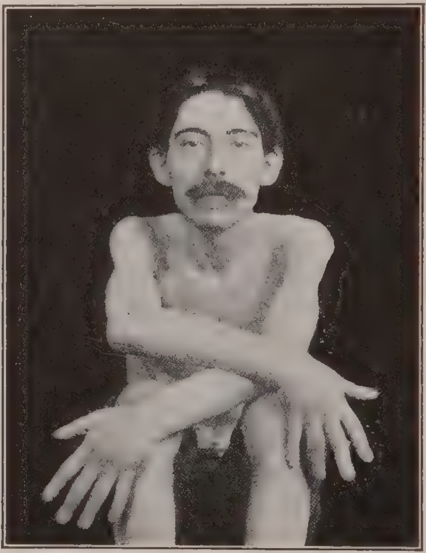

FIG. 38.—Hypertrophic Pulmonary Osteo-arthropathy. (Thayer.)

3. *Acromegalia* (see page 8) affects chiefly the bones and soft tissues of the hand. The x-ray picture is characteristic.

4. *Hypertrophic, atrophic, or tuberculous disease* of the wrist-joint will be described below (see Examinations of the joints, page 481).

5. "*Weeping sinew*" or "ganglion" (tenosynovitis) forms a fluctuating, spindle-shaped swelling along one of the tendons of the wrist, slow and almost painless in its course. It may be tuberculous, in which case the sac is generally divided into several parts ("compound ganglion"); tubercle bacilli may occasionally be demonstrated in the exudate.

6. *Neoplasms* (see Fig. 37).

THE HANDS

I. Evidence of Occupation

The horny, stiffened hands of the "son of toil," the stains of paint in house painters, the flattened, calloused finger-tips of the violinist, the worn fingers of the sewing woman, afford us items of information which are sometimes useful and worth a rapid glance in routine examination.

II. Temperature and Moisture

(*a*) The *cold, moist hand* is most commonly felt in "nervous" people under forty. It is almost never seen in heart disease, which its possessor often fears, and does not mean "poor circulation," but vasomotor disturbances of neurotic origin.

(*b*) *Cold, dry extremities*—hands, feet, nose, ears—may mean simply fatigue, exposure to low temperature, or insufficient exercise; but in the course of chronic disease they usually mean weakness of the heart, and hence are serious.

(*c*) *Warm, moist hands* are felt in *thyrotoxicosis*, and if the warmth and moisture are present most of the time this disease is strongly suggested, and a search for tremor, rapid heart, goitre, and bulging eyes should be made.

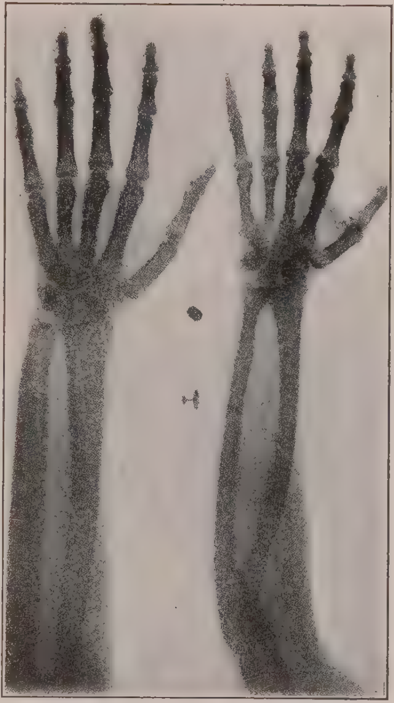

Fig. 39.—Radiographs of the Hand and Arm of a Case of Hypertrophic Pulmonary Osteoarthropathy (the left figure) compared with the hand and arm of a normal individual of the same height (the right figure). Note especially the thickening of the radius and ulna. (Thayer.)

(*d*) The hot, dry hand ordinarily means fever, as temperature record will demonstrate.

III. Movements of the Hands

(*a*) The *manner of shaking hands* gives us vague but useful impressions of the patient's temperament. The nervous, cramped, half-opened hand, which never really grasps and gets away as soon as possible; the firm, hearty grasp; the limp, "wilted" hand—furnish hints of character that every physician must take account of.

In fevers or toxæmic states (typhoid, alcoholism) there are two sets of movements which recur so often that names have been given them, viz.: 1. *Carphologia*—picking and fumbling at the bed clothes. 2. *Subsultus tendinum*—involuntary twitching and jerking of the tendons in the wrist and on the back of the hand, usually associated with tremor and carphologia.

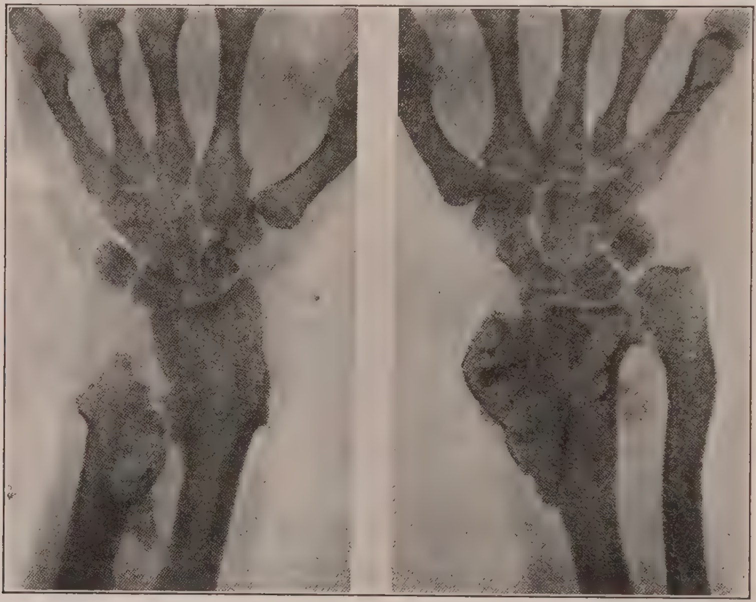

Fig. 40.—Radiograph of the Wrists in Hypertrophic Pulmonary Osteo-arthropathy. (v. Ziemssen's Atlas.)

(*b*) *Tremor of the Hands.*—To test for ordinary tremor, we ask the patient to extend and separate his fingers widely. The motions are then apparent.

Causes: 1. Nervousness, cold, or old age. 2. Fever and toxæmia. 3. Alcohol (less often lead, tobacco, morphine, or other drugs). 4. Graves' disease. 5. Paralysis agitans. 6. Multiple sclerosis. 7. Hysteria.

Most of these tremors need no comment. The *intention tremor* of multiple sclerosis (sometimes seen also in hysteria) is often exag gerated into coarse shaking movements when the patient tries to pick up a pin, drink a glass of water, or do any other act calling for the volitional coordination of the small hand muscles. In the presence of such a tremor we should look for *nystagmus* (see above, page 17), a spastic gait (see page 497), and a slow, staccato speech. This group of symptoms suggests multiple (or insular) sclerosis.

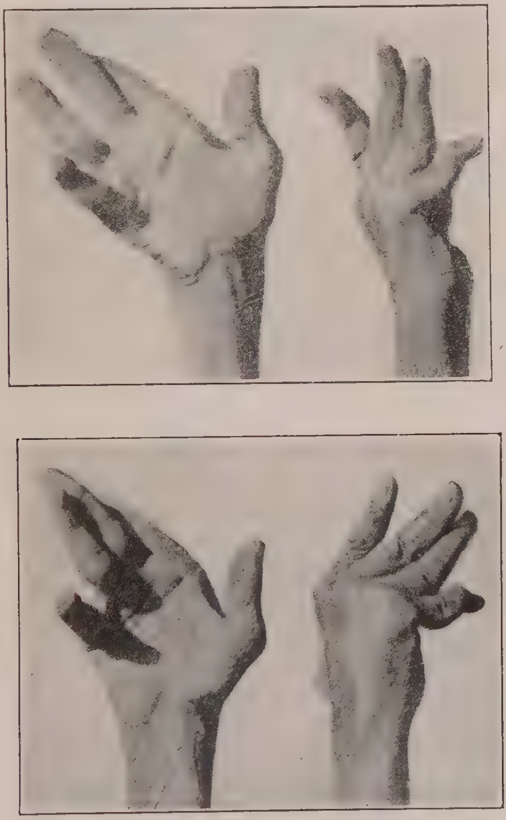

Fig. 41.—Athetosis. Successive positions of the hands. (Curschmann.)

In direct contrast with this is the *pill-rolling tremor* of paralysis agitans, which usually *ceases during voluntary movements*. The thumb and forefinger are near or touch one another, and move as if they were rolling a bread-pill. This tremor is usually associated with an immovable, expressionless face, a stiffened neck and back, and a peculiar attitude and gait (see below, page 498).

The other varieties of tremor can usually be recognized by the history and associated symptoms.

(*c*) *Spasms or coarse twitchings of the hand* due to:

1. *Jacksonian epilepsy*—convulsive attacks which begin in and may remain confined to one set of muscles, often preceded by prickling or other paræsthesia of the part affected, but *without loss of consciousness.* These muscle spasms are due usually to an irritation of the corresponding motor area in the cortex cerebri (tumor, softening, chronic meningitis, etc.), but may also occur in uræmia and diementia paralytica. Coma and general spasms may follow.

2. *Professional Spasm.*—Writers, violin-players, and others who use one set of muscles continually are often attacked with *painful cramps* in the muscles used ("writer's cramp"). Weakness and pain are usually far more prominent than spasm.

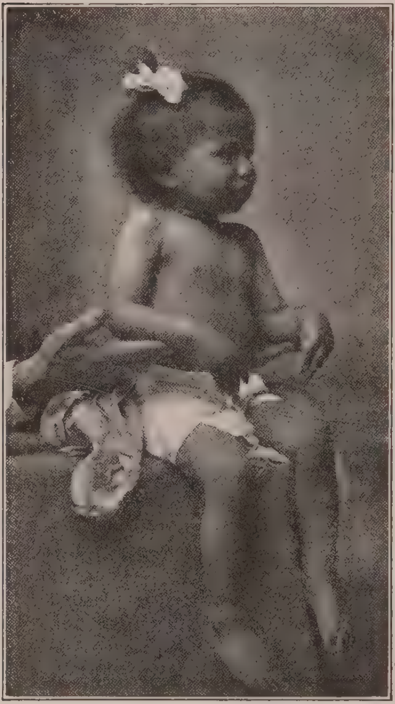

Fig. 42.—Tetany. (Masland.)

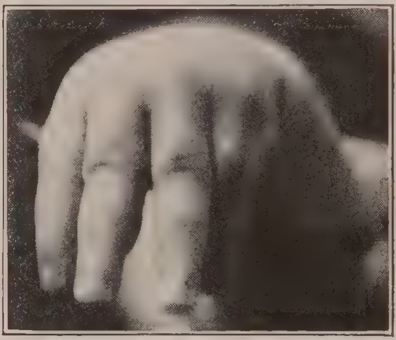

Fig. 43.—Tetany.

3. *Chorea and Choreiform Movements.*—True, acute, infectious chorea (Sydenham's) occurs chiefly in children between five and fifteen, generally in those who have joint troubles or heart disease, and ends in eight or ten weeks. The hands are usually affected first, and their movements are like those of restlessness and are quasi-purposive, *i.e.*, movements that might have been made intentionally, though they

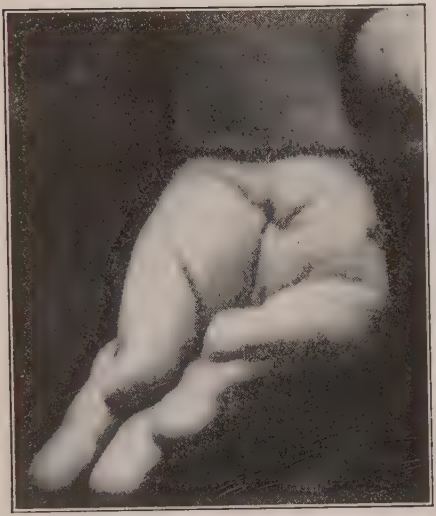

Fig. 44.—Tetany.

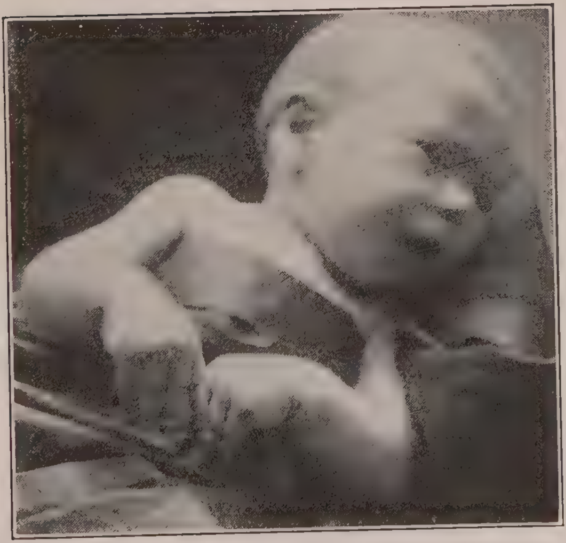

Fig. 45.—Tetany.

are not. At first sight one would surely think the child was simply fidgety.

Similar movements occur in *pregnant women* or sometimes after parturition, but the type is much severer and is apt to be associated with maniacal symptoms.

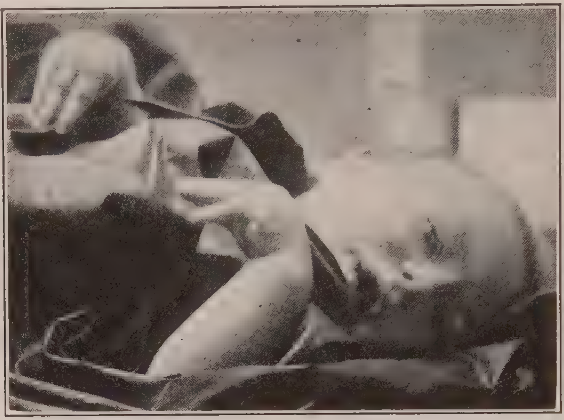

FIG. 46.—Tetany.

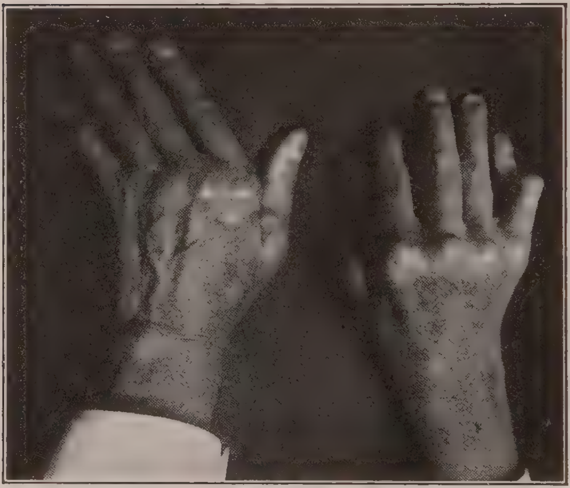

FIG. 47.—Atrophic Arthritis with "Flipper Hand."

Habit spasms or tics are much commoner in the face, throat and shoulders but also reach the hands occasionally. They constitute an independent chronic neurosis and may or may not be associated with mental or emotional disturbances. Winking and nodding movements are commonest. They have no relation to infectious chorea, to the joints or the heart.

Post-hemiplegic chorea refers to similar movements in the paralyzed hands of hemiplegic cases (children or adults). True infectious chorea may also affect only one-half the body (hemichorea).

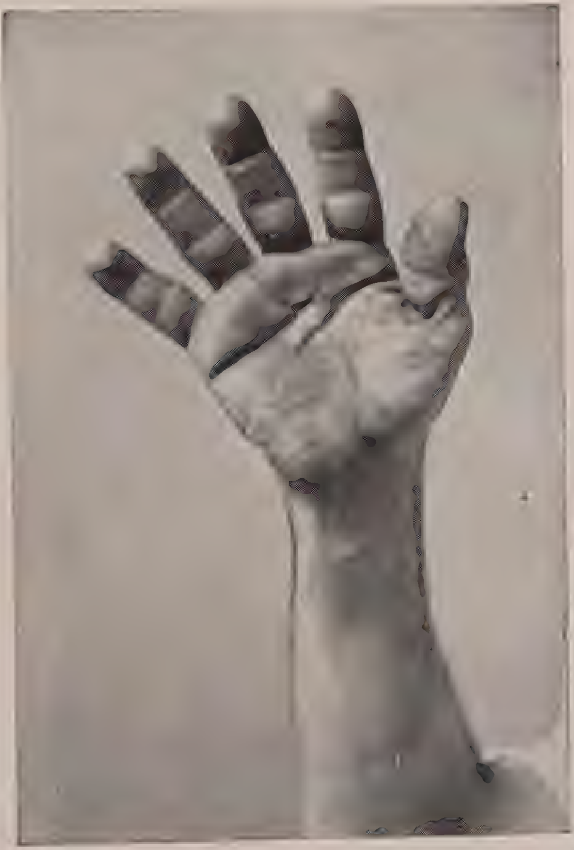

Fig. 48.—Spade Hand in Myxœdema.

In *hysteria* or by a short of *psychic contagion* similar movements are sometimes taken up in schools and institutions, and last till their cause is understood and removed.

Chronic choreiform movements occur also in the rarer congenital forms of paralysis with or without idiocy.

4. *Athetosis* (see Fig. 41) means slow twisting and bending movements of the fingers, quite involuntary and always secondary to organic cerebral lesions (hemiplegia, infantile cerebral paralysis).

5. *Tetany* (see Figs. 42, 43, 44, 45 and 46)—a peculiar spasm of the hands (often of the feet as well), occurring in the course of diseases involving obstruction of the pylorus, in children, in nursing women, after gastric lavage, and after thyroidectomy,[1] usually lasting minutes or hours—rarely days. The gastric or obstructive type of tetany (with or without uremia) seems to depend on the modifications of the gastric acidity and so of the haemic alkalinity.

IV. Deformities of the Hands

1. "*Claw hand*" results from paralysis of the interossei and lumbricales with contractures, and occurs when the median or ulnar nerves are paralyzed, and in progressive muscular atrophy, syringomyelia, and chronic poliomyelitis.

2. "*Flipper hand*" (see Fig. 47), a common result of the contractures in late cases of atrophic arthritis. Other deformities of the fingers are common in this disease and in gout.

3. "*Hemiplegic hand*," a result of the contractures following hemiplegia from any cause.

4. *Myxœdema* results in thickening and coarsening of the tissues of the hand ("spade hand") without bony enlargement; but the spade hand is a fairly common type without myxœdema, and one needs to see it rapidly develop in connection with other myxœdematous lesions before it can have diagnostic significance. (The same is true of the myxœdematous face.) (See Fig. 48.)

5. *Acromegalia* produces general enlargement of the bones and other tissues of the hands and feet.

6. *Pulmonary Osteo-athropathy.*—Any long-standing disease of the heart, lungs, or pleura, and any abdominal tumor (liver, spleen) which elevates the diaphragm and reduces lung capacity, may be followed by this peculiar hypertrophic change in all the tissues of the extremities. Mild forms produce "*clubbed fingers*," a bulbous enlargement of the finger-tips with double curvation of the nails, lateral and antero-posterior[1] (see Fig. 51). In severer forms the bones of the hand and wrist are also considerably enlarged (see Figs. 38 and 40).

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[1] When the parathyroid glands are accidentally removed.

[1] Clubbed fingers are occasionally seen in a variety of other diseases: *e.g.*, hepatic abscess, nephritis, congenital syphilis; and even in apparently healthy persons.

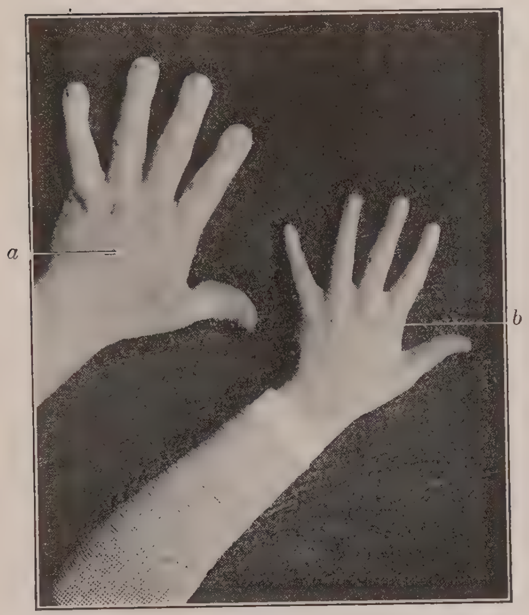

FIG. 49.—*a*, Acromegalic Hand. *b*, Normal Hand.

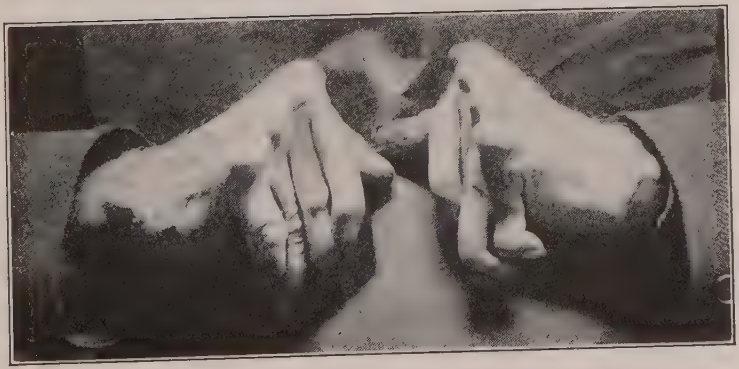

FIG. 50.—Atrophic Arthritis.

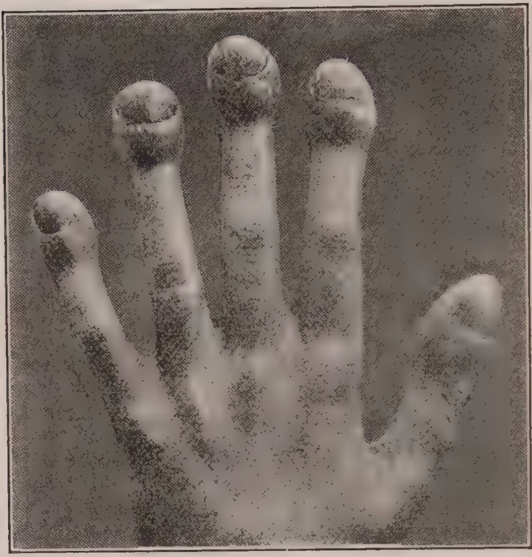

FIG. 51.—Clubbed Fingers.

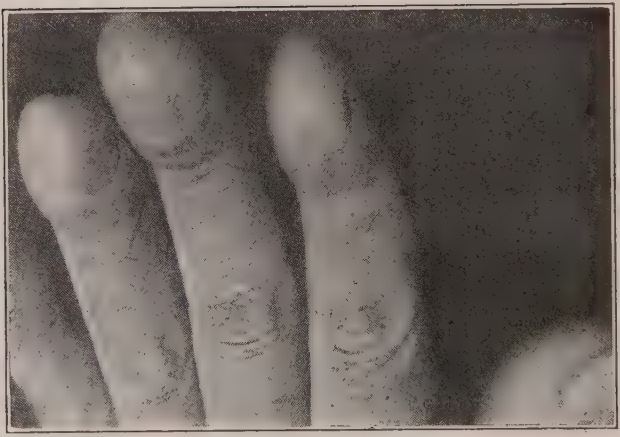

FIG. 52.—Clubbed Fingers.

7. *Heberden's nodes*, later described under the head of hypertrophic arthritis, are here pictured (Fig. 54). The distinction from *gout* will later be referred to (page 482).

8. *Atrophic arthritis* (Fig. 47) (further described on page 488) presents its most typical lesions in the hands and wrists. The constriction line opposite the articulation is observed in late cases, but ordinarily multiple spindle-joints symmetrically arranged are all that we see. The boggy feel, the trophic disturbances, and the chronic course are usually diagnostic; but x-ray examination is necessary to establish the diagnosis which is important because of the unfavorable prognosis which it involves.

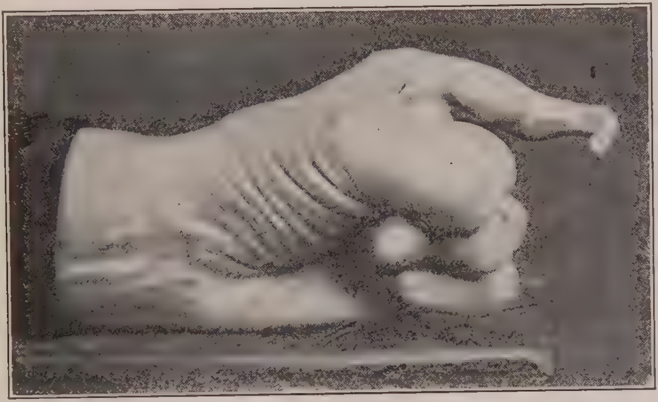

Fig. 53.—Raynaud's Disease.

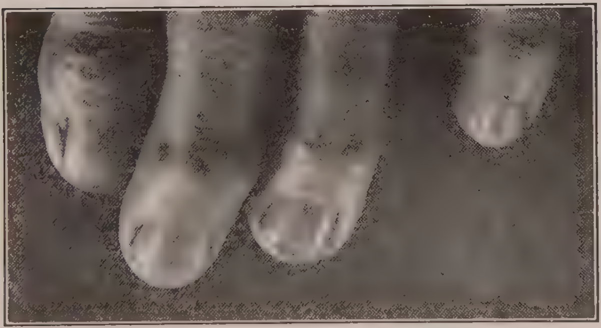

Fig. 54.—Heberden's Nodes.

9. *Syphilitic and tuberculous dactylitis* (see Fig. 55), seen as a rule in young children, are not distinguished from each other by the physical signs. Diagnosis rests upon the history, the course, the *x*-ray, the Wassermann reaction, the results of giving tuberculin or Salvarsan, and the evidence of syphilitic or tuberculous lesions elsewhere. In either disease we have a chronic, almost painless, boggy, red enlargement of one phalanx, or more, due to an indolent inflammation which starts from the bone or periosteum and usually burrows to the surface, to produce a chronic discharging sinus or ulcer.

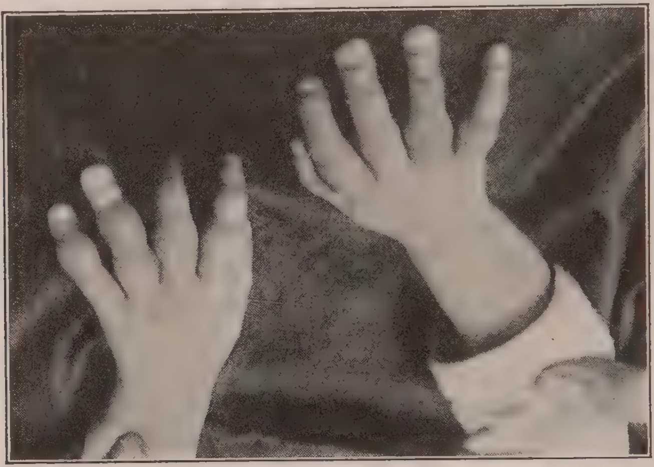

FIG. 55.—Tuberculous Dactylitis.

10. *Raynaud's disease* attacks the fingers more often than any other part. Osler distinguishes three grades of intensity: A Local *syncope* ("dead fingers") following exposures to slight cold or emotional strain. The fingers become white and cold. The condition usually passes off in an hour or two. From similar causes we may have: B. Local *asphyxia* ("chilblains"), producing congestion and swelling with or without pain and stiffness and with heat or coldness of the part. C. Local or symmetrical *gangrene*. If local asphyxia persists, gangrene results. (See also under Erythromelalgia, p. 459.)

11. *Morvan's Disease.*—As a part of syringomyelia multiple arthropathies (atrophic arthritis) and painless felons may develop in the hands (see Fig. 56). The appearances may strongly suggest:

12. *Leprosy,* in which there is likewise anæsthetic necrosis of phalanges; but the two diseases can usually be distinguished by a study of the lesions and symptoms in other parts of the body.

13. *Dupuytren's contraction of the palmar fascia* is commonest in adult men, and gradually produces a permanent, painless flexion of the little finger in one or both hands. A tense band is felt in the palm. The ring finger may also be affected; less often the others. It burn and felon are excluded, the diagnosis is obvious.

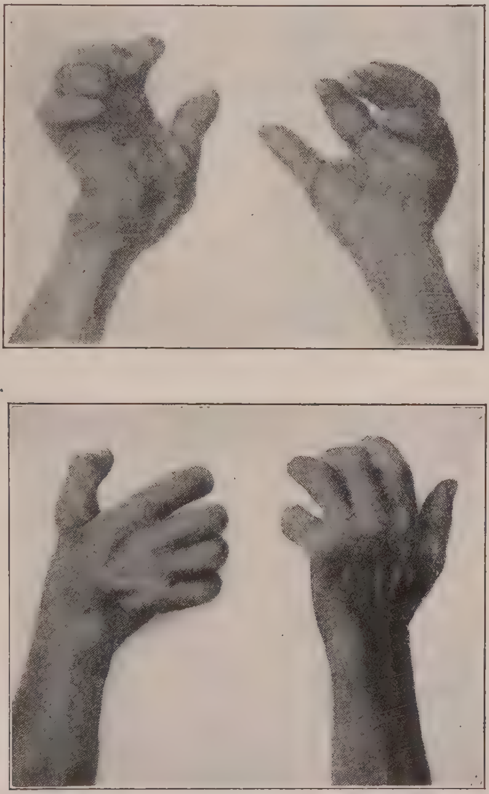

FIG. 56.—Morvan's Disease.

THE NAILS AND FINGER TIPS

1. The *nutrition of the nails* suffers in chronic skin diseases, in myxœdema, in many nerve lesions (neuritis, hemiplegia, syringomyelia, etc.), dementia paralytica; also in atrophic arthritis.

2. A *transverse ridge and groove* on the nails often form when their growth is resumed after an acute illness. The movement of this ridge from the matrix to the free edge is said to take about six months (see Fig. 57).

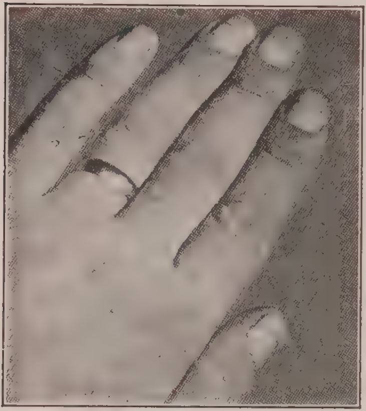

FIG. 57.—Grooved Nails after Acute Illness.

3. *Hang-nails* possess a certain medical interest, because in some individuals they become sore when the general condition is below par, and constitute a rough index of the degree of resistance to infection. They may become infected and lead on to suppuration (*paronychia*).

4. *Indolent sores* around the nail should rouse the suspicion of tuberculosis or syphilis, especially in a child.

5. (*a*) *Cyanosis*, the slatey or purplish-blue color of venous congestion, can be well seen in the nails. (*b*) *Anæmia*, if well marked, blanches the tint of the tissues seen through the nail, but the diagnosis should invariably be confirmed by a hæmoglobin estimate.

6. *Incurvation* of the nails has already been referred to as a part of the condition known as "clubbed fingers" (page 54).

7. *Capillary pulse* (see below, page 91).

8. *Tender finger ends* not infrequently occur in septic endocarditis and may help in the diagnosis of that disease. Minute ecchymoses are occasionally present as well. Both phenomena are, I suppose, embolic.

THE BACK

The evidences of spinal tuberculosis, spinal curvature, and of the spinal form of infectious and of hypertrophic arthritis will be described later (page 480 *et eeq.*).

I. Stiff Back

"Stiff back" may be due not only to the joint troubles just mentioned, but also and more commonly to *lumbago*, a painful affection of the lumbar muscles without known pathologic basis. Clinically it is characterized by pain when the muscles are used, as in bending forward to tie one's shoes and in recovering the upright position. There is no bony soreness, no involvement of the sacro-iliac joints, and sideways bending is usually freer than in hypertrophic arthritis. The pain of lumbago does not radiate around the chest or down the legs, and is not especially aggravated by coughing or sneezing, but it sometimes extends down low into the fascia of the lumbar muscles over the sacrum. The age of the patient (usually over thirty) distinguishes most cases of lumbago from spinal tuberculosis. "Stiff neck" often accompanies or precedes it and some relation to meteoric conditions can often be traced. The disease is self limited and should end in a few days or at most a few weeks. Cases of longer duration are probably due to infectious or hypertrophic *spinal arthritis* or to *tuberculosis*.

Metastatic cancer of the vertebræ often follows cancer of the breast, producing a stiff, painful spine. The *x*-ray picture is usually characteristic.

II. Sacro-iliac Disease

Tuberculosis of this joint has long been known and calls attention to its presence by pain, psoas spasm, and a limp. If the wings of the ilium are forcibly pressed together, the pain in the joint is much increased. Abscess formation is often the first distinctive sign. The motions at the hip-joint are not restricted and the local signs of vertebral caries are absent. The duration of the disease, the local reaction after tuberculin injection and the formation of abscess distinguish it from other lesions of the sacro-iliac joint.

Goldthwait[1] has shown that the sacro-iliac joint is subject to most of the diseases of other joints, and that some (*e.g.*, hypertrophic arthritis) are not at all uncommon there. Many of the pains in the back complained of by women during menstruation or in pelvic disorders are referred precisely to the sacro-iliac articulation and are probably due to lesions of that joint. Many cases diagnosed as "lumbago" are probably due to one or another sacro-iliac lesion, strain, sprain or subluxation. The diagnostic points are—on the positive side: (*a*) Pain or tenderness directly over the joint. Such *pain* may be *elicited by raising the leg while the knee is kept stiff*. It is also referred in many cases to the course of the sciatic nerve so that *many, perhaps most, cases of so-called sciatica are due in fact to sacro-iliac disease.* It is often worse at night. (*b*) Abnormal mobility of the sacro-iliac joint. (*c*) A tendency to lean the trunk away from the affected side when standing. (*d*) Limitation of lateral bending of the spine to one side or the other when the patient stands with the knees stiff.

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[1] Goldthwait: Boston Medical and Surgical Journal, March 9th, 1905.

On the negative side the absence of limitation in the motions at the hip-joint, the negative *x*-ray, the free forward bending (when the patient sits during the test), the absence of fever, leucocytosis and abscess formation are important.

A strong nervous element is present in many cases.

III. Spinal Curvatures

Diagnosis is not difficult, provided we are led to examine the back at all.

(*a*) *Kyphosis* or *backward* convexity of the spine, if sharply angular usually means Pott's disease (tuberculosis). If the curve is gentle and gradual it may be due to "*round shoulders*," to *hypertrophic arthritis*, to *barrel chest*, Paget's disease, or rickets. The rachitic curve is flaccid, is due simply to muscular weakness, and is associated with other evidences of rickets. In barrel chest and Paget's disease the kyphosis goes with the other signs of those diseases. In hypertrophic arthritis the curve is rigid, irreducible, and usually painless. "Round shoulders" can be straightened by muscular exertion, and represent a habit of posture.

(*b*) *Lordosis*, an exaggeration of the normal forward convexity of the lumbar spine, is seen in tuberculosis of the hip or spine, in paralysis of the dorsal or abdominal muscles (especially muscular dystrophy), and in abdominal tumors (pregnancy), which need to be counterbalanced by backward bending.

(*c*) *Scoliosis* is a combination of lateral curvature with twisting of the spine. In slight or doubtful cases the tips of the spinous processes should be marked with a colored pencil, which makes the deviation easily visible. Severe cases cannot be mistaken.

IV. Tumors of the Back

(*a*) *Aneurism* of the descending aorta may point in the back near the angle of the left scapula (see below, page 263). It is the only pulsating tumor of this region.

(*b*) *Perinephritic abscess* usually points between the crest of the ilium and the twelfth rib, a few inches from the spine (see page 410).

(*c*) *Tuberculous abscess* ("cold abscess"), originating in vertebral tuberculosis, may point in the same region, though more often it follows down the sheath of the psoas and points near Poupart's ligament. "Cold abscess," starting from a necrosed rib, is often seen in the back. The probe leads to dead bone at the end of the sinus. Microscopic examination of excised pieces is the only way of excluding *actinomycosis*, though this disease is less apt to form sinuses.

(*d*) *Sarcoma of the scapula*, the only tumor of the scapula that is often seen, occurs in children and rarely after the second decade. With a solid, nearly painless tumor of this bone in a child, sarcoma should always be suspected. Benign exostoses are possible, but usually occur later in life. Histological examination will decide.

(*e*) *Epithelioma*, arising from the skin of the back, presents the ordinary evidences of this form of cancer.

(*f*) The multiple subcutaneous abscesses due to *glanders* ("*farcy buds*") are more common on the extremities, but may be found on the trunk as well. Flattened, oval fluctuating nodes with slight tenderness are suggestive. Bacteriological examination of the purulent contents settles the diagnosis.

V. Prominent Scapula

This is due usually to:

(*a*) *Lateral curvature* of the spine (see above).

(*b*) *Serratus paralysis*, recognized by the startling prominence of the scapula if the patient pushes forward with both hands against resistance ("angel-wing" scapula).

VI. Scaphoid Scapula

In congenital syphilis and some non-syphilitic conditions the median or vertebral border of the scapula is sometimes markedly *concave*.

VII. Spina Bifida

A congenital, saccular tumor, connecting through a bony defect with the interior of the spinal canal at any point between the occiput and the sacrum; nine-tenths of all cases occur in the lowest third of the spinal column. There is no other congenital tumor in this position communicating with the spinal canal.

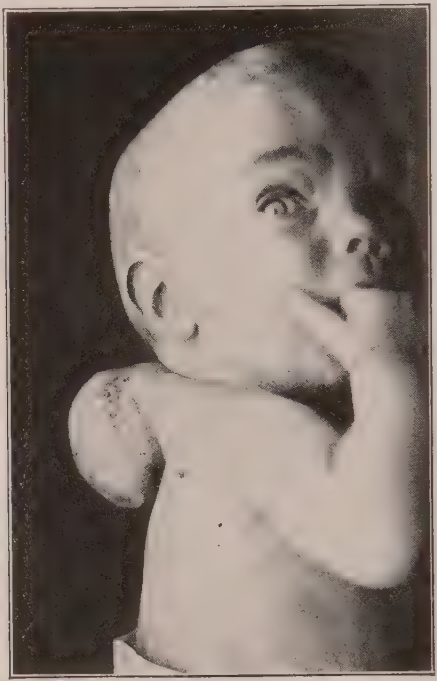

FIG. 58.—Spina Bifida with Meningocele.

In the sacral region there are other congenital sinuses, tumors, dermoid cysts, lipomata, and others. Their nature can be learned only by incision. It is important to settle their relation to spina bifida by the presence or lack of communication with the spinal canal.

CHAPTER IV

THE CHEST
TECHNIQUE AND GENERAL DIAGNOSIS

INTRODUCTION

I. Methods of Examining the Thoracic Organs

To carry out a thorough examination of the chest we do five things: 1. We look at it; technically called "inspection." 2. We feel of it; technically called "palpation." 3. We listen to the sounds produced by striking it; technically called "percussion." 4. We listen to the sounds produced within it by physiological or pathological processes; technically called "auscultation." 5. We study pictures thrown on the fluoroscopic screen or on a photographic plate by the Roentgen rays as they traverse the chest; technically called "radioscopy."

Measuring the dimensions or movements of the chest ("mensuration") and the spirometric measurement of "vital capacity" are often mentioned as co-ordinate with the above methods, but yield very little information of practical value.

Without some knowledge of the regional anatomy of the chest no intelligent investigation of the condition of the thoracic organs can be carried on. Accordingly, I shall begin by recalling very briefly some of the most essential anatomical relations.

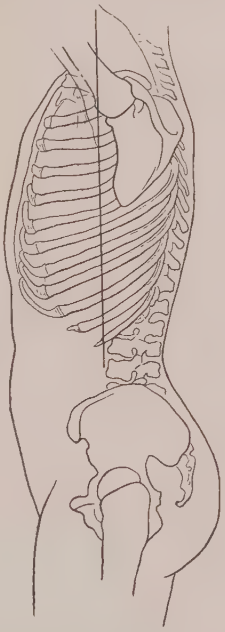

Fig. 59.—The Mid-axillary Line.

II. Regional Anatomy of the Chest

It seems to me a mistake to divide the chest into arbitrary portions and to describe physical signs with reference to such division. The seat of any lesion can best be described by giving its relation to the clavicle, sternum, or ribs on the front and sides of the chest, and to the scapulæ and ribs behind. Thus we may speak of râles as heard "above the left clavicle in front," "below the right scapula behind," "between the seventh and ninth ribs in the axilla," and so on. When we want to state more exactly what part of the axilla anteroposteriorly is affected we may refer to the "mid-axillary line" (see Fig. 59); or better, we may place the lesion by measuring the number of centimetres or inches from the median line of the sternum. In a similar way the place of the apex impulse of the heart (whether in the normal situation or farther toward the axilla) can be determined by measuring from the median line of the sternum. Measurements referring to the nipple are useless in women with relaxed or hypertrophied breasts. But as a general rule they convey more useful and reliable information than measurements from mid-sternum.

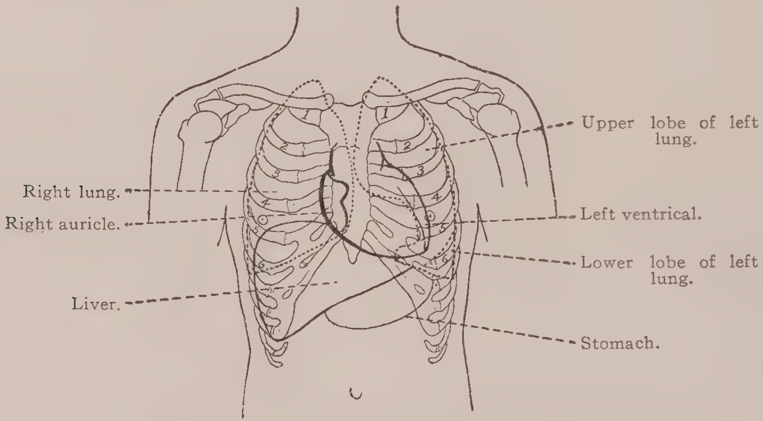

FIG. 60.—Position of the Heart, Lungs, Liver and Stomach. The dotted lines correspond to the outlines of the lung; the heavy continuous line represents the heart; while the position of the liver and of the lower border of the stomach is indicated by light continuous lines. The ribs are numbered.

If, then, we confine ourselves chiefly to the bones of the chest as landmarks, and fix, with reference to them, the position of any portion of the internal organs which we desire to study, it becomes unnecessary to memorize any technical terms or to learn the position of any arbitrary lines and divisions such as are frequently forced upon the student. The only points which it is necessary to memorize once for all are:

1. The position of the heart, lungs, liver, and spleen with reference to the bones of the chest.

2. The position of certain points which experience has taught us have a certain value in physical diagnosis. I mean (*a*) the so-called

"valve areas" of the heart, which do not correspond to the actual position of the valves, for reasons to be explained later on, and (*b*) the percussion outlines of the heart, liver, and spleen. These outlines do not correspond in size with the actual dimensions of the organs within, yet there is a definite relation between the two which remains relatively constant, so that we can infer the size of the organ itself from the outlines which we determine by percussion. The position of the organs themselves is shown in Figs. 60, 61, and 62. It will be noticed in Fig. 58 that the lungs extend up above the clavicles and overlap the liver and the heart—facts of considerable importance in the physical examination of these organs, as will be later seen. It is also to be noticed how small a portion of the stomach is directly accessible to physical examination, the larger part of it lying behind the ribs and covered by the liver. The normal pancreas and kidneys are practically inaccessible to physical examination.

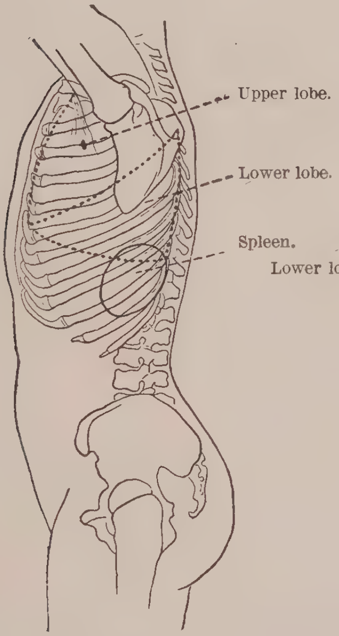

FIG. 61.—Position of the Left Lung from the Side, and of the Spleen.

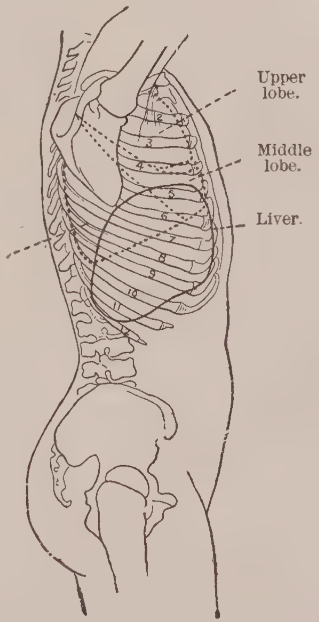

FIG. 62.—Position of the Right Lung from the Side, and of the Liver.

The percussion outlines—corresponding to those portions of the heart, liver, and spleen which lie immediately beneath the chest walls—will be illustrated in the section on Percussion (see page 125).

INSPECTION

Much may be learned by a careful inspection of all parts of the chest, but only in case the clothes are wholly removed. A good light is essential, but this does not always mean a *direct* light; for example, when examining the front of the chest it is often better to have the patient stand with his side to the window so that the light strikes obliquely across the chest, accenting every depression and making every pulsation a moving shadow. In searching for abnormal pulsations, this oblique light is especially important.

In examining the thorax we look for the following points:

1. The size.
2. The general shape and nutrition.
3. Local deformities or tumors.
4. The respiratory movements of the chest walls.
5. The respiratory movements of the diaphragm.
6. The normal cardiac movements.
7. Abnormal pulsations (arterial, venous or capillary).
8. The peripheral vessels.
9. The color and conditions of the skin and mucous membranes.
10. The presence or absence of glandular enlargement.

I. Size

Small chests are seen in patients who have been long in bed from whatever cause; also in those who have suffered in infancy from rickets, adenoid growths in the naso-pharynx, or a combination of the two diseases. Abnormally large chests ("barrel chest") are seen chiefly in elderly people with stiff cartilages. Of course the chests of healthy individuals vary a great deal in size at any given age, and I refer only to variations greater than those normally found.

II. Shape

There are marked differences in shape between the child's and the adult's chest in health. A child's trunk, as compared with that of an adult, is far more nearly cylindrical; that is, the anteroposterior diameter is nearly as great as the lateral. The adult's chest is at first flattened from before backward. Later it returns more nearly to the childish shape as cartilages stiffen ("barrel chest").

In childhood the commonest pathological modifications are due to adenoids or to rickets; in middle and later life to calcified cartilages, to phthisis, or old pleuritic disease.

1. *The Rachitic Chest*

The sternum generally projects ("*pigeon breast*"), but in some cases, especially when rickets is combined with adenoid hypertrophy, there may be a depression at the root of the sternum resulting in the condition known as "*funnel breast*"[1] (Fig. 63). The sides of the chest are compressed laterally and slope in to meet the sternum as the sides of a ship slope down to meet the keel (*pectus carinatum*) (Figs. 65 and 66). From the origin of the ensiform cartilage a depression or groove is to be seen running downward and outward to the axilla and corresponding nearly to the attachment of the diaphragm. This is sometimes spoken of as "*Harrison's groove.*" The *lower margin of the ribs* in front often *flares out*, owing to the enlargement of the liver and spleen below and the pull of the diaphragm above. Along the line of the chondro-costal articulation there is to be felt, and sometimes seen, a line of eminences or swellings, to which the name of "*rachitic rosary*" has been given.

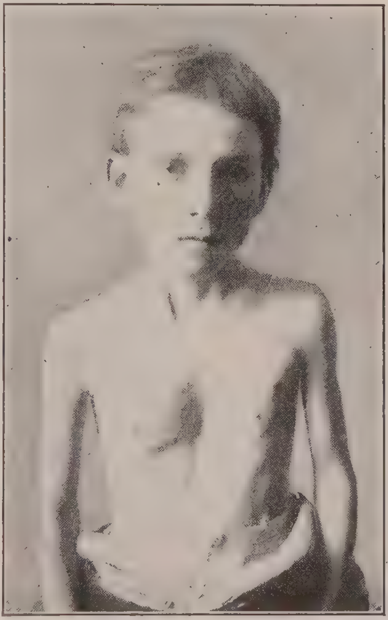

Fig. 63.—Funnel Breast.

2. *The "Paralytic Thorax"*

Fig. 67 conveys a better idea of this form of chest than any description. The normal anteroposterior flattening is exaggerated so that such persons are spoken of as "*flat-chested.*" The clavicles are very prominent, owing to falling in of the tissues above and below them; the shoulders are stooping, the scapulæ prominent, and the neck is generally long. The angle where the ribs meet at the ensiform cartilage, the so-called "*costal angle,*" is in such cases very sharp. This type of chest has often been supposed to be characteristic of phthisis, but may be found in persons with perfectly healthy lungs. On the other hand, phthisis frequently exists in persons with normally shaped chests or with abnormally deep chests (Woods Hutchinson). (See Fig. 178, page 299.)

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[1] In some cases this condition appears to be congenital.

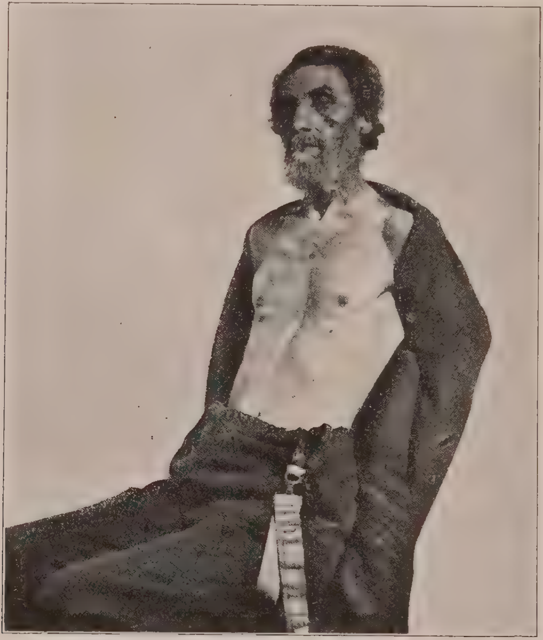

FIG. 64.—Acquired Depression at the Root of the Ensiform Cartilage. The patient is a shoemaker of seventy, who has all his life pressed against his breast bone the shoe on which he worked.

3. *The "Barrel Chest"*

Nothing is less like a barrel than the "*barrel chest.*" It represents an extreme degree of the physiological enlargement of the thorax seen so often after middle life, especially in males. Its most striking characteristic is its greatly increased anteroposterior diameter, so that it approaches the form of the infant's chest. The costal angle is very obtuse, the shoulders are high, and the neck is short. The respiratory movements of the barrel chest will be spoken of later (see Figs. 68 and 69).

(a) *Nutrition of the Chest Walls*

Emaciation is readily appreciated by inspection. The ribs are unusually prominent, the scapulæ stand out, and the clavicles project.

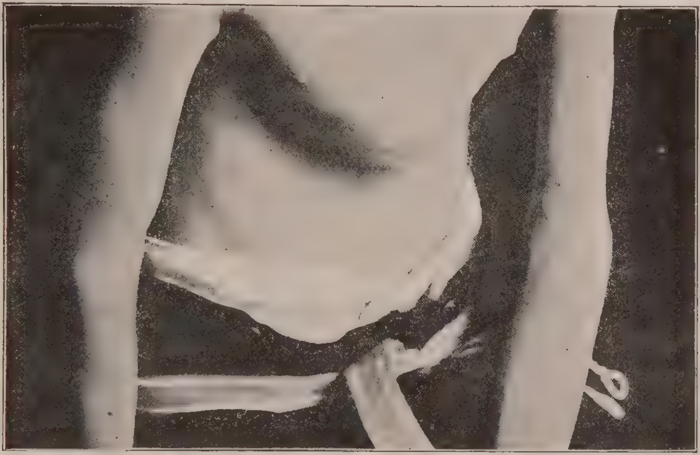

Fig. 65.—Pigeon Breast.

All this may be seen independently of any change in the shape of the chest such as was described above under the title of Paralytic Thorax. Tuberculosis of the apices of the lungs may produce a marked falling in of the tissues above and below the clavicle independent of any emaciation of the chest itself.

III. Deformities

The abnormalities just enumerated are symmetrical and affect the whole thorax. Under the head of Deformities, I shall consider chiefly such abnormalities as affect particular portions of the chest and not the thorax as a whole.

1. *Spinal Curvatures and Twists*[1]

Slight degrees of deformity are best seen by marking with a skin-pencil the position of the spinous processes (see Fig. 71). The more marked cases of lateral curvature, which are usually accompanied by a certain amount of *twisting*, give rise to considerable displacement of the thoracic organs and render unreliable the usual bony landmarks, with reference to which we judge of the position of the intrathoracic organs. By such deformities the apex of the heart may be pushed up into the fourth space or out into the axilla, or portions of the lungs may be compressed and made atelectatic. The bulging on the convex side of the curve may simulate an aneurismal tumor. Pott's disease of the spine should be looked for as a part of the routine inspection of the chest. It is sometimes better felt than seen.

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[1] See also page 60. The lesions are referred to here only in relation to their effects on heart and lungs.

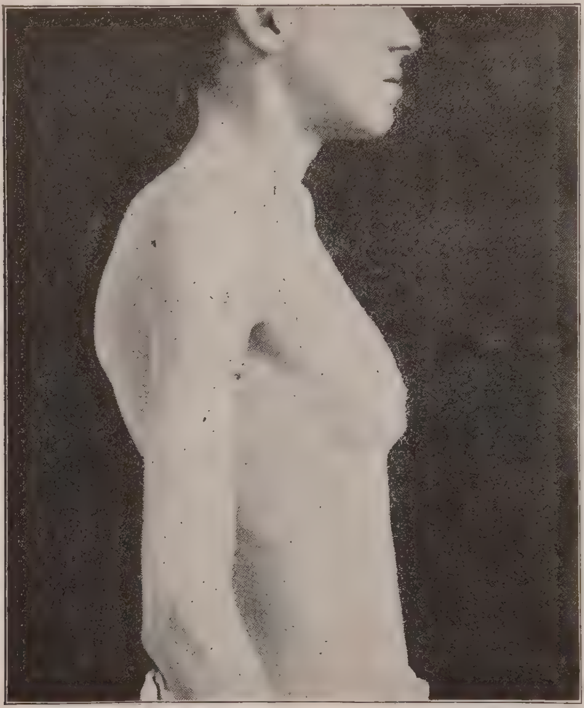

Fig. 66.—Pigeon Breast.

2. *Flattening of One Side of the Chest*

In chronic phthisis, cirrhosis of the lung, or long-standing pleurisy (serous, fibrous or purulent), marked falling in of one side of the chest is often to be seen (see Figs. 67 and 73). The shrinkage of the affected side is made more obvious by contrast with the compensatory hypertrophy of the sound lung, which makes the sound side unusually full and prominent.

3. *Prominence of One Side of the Chest*

In pneumothorax or pleural effusions, and sometimes in malignant disease of the lung or pleura, there is a marked increase in the size of the affected side of the chest. In pneumothorax or pleuritic effusion we usually see, in addition to the above enlargement of the affected side, a smoothing out of the intercostal depressions so that the surface of that side is much more uniform than the other side. Bulging of the interspaces from great pressure within the chest rarely occurs. I have never seen it.

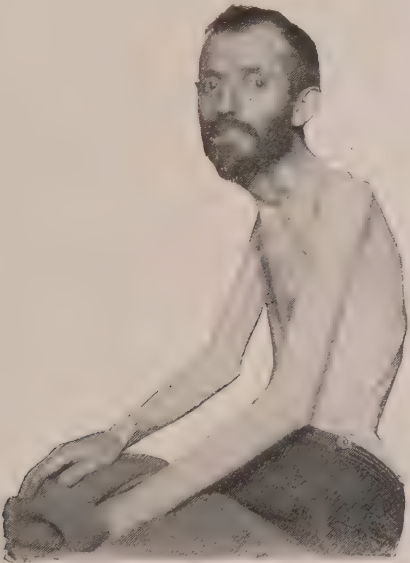

Fig. 67.—The Paralytic Thorax.

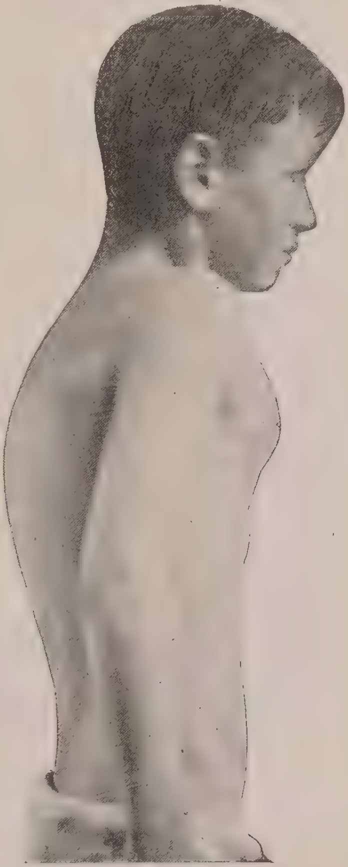

Fig. 68.—Barrel Chest in a Case of Bronchial Asthma (æt. 13).

4. *Local Prominences*

In nearly one-quarter of all healthy chests that part of the thoracic wall which overlies the heart (the so-called "precordial region") is abnormally prominent. The cause of this condition is much disputed. A similar prominence may be brought about in children (whose thoracic bones are very flexible) and occasionally in older patients, by the outward pressure of an enlarged heart or of an effusion in the pericardial sac. The prominences due to spinal curvature have been already mentioned. Less common causes of local prominence are:

1. *Aneurism* of the arch of the aorta.
2. *Tumor* of the chest wall (lipoma, sarcoma, gumma), of the lung, mediastinum, or of the thoracic glands pressing their way outward.
3. "*Cold abscess*" (tuberculosis, actinomycosis) of a rib or of the sternum.
4. *Empyema* perforating the chest wall, the so-called "empyema necessitatis."

IV. THE RESPIRATORY MOVEMENTS

1. *Normal Respiration*

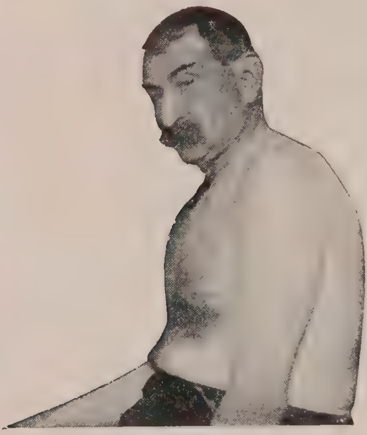

FIG. 69.—Barrel Chest.

During normal respiration, one sees the ribs move outward and upward with inspiration, and downward and inward with expiration. Possibly one catches some hint of the movements of the diaphragm at the epigastrium. In men, diaphragmatic breathing is more marked, while in women breathing is mostly of the "costal type;" that is, is done by the intercostal muscles. In certain diseases an exaggeration of the costal or of the diaphragmatic type of breathing may be seen. When costal cartilages become ossified, the ribs move little, and most of the work of respiration is performed by the diaphragm, whose pull upon the lower ribs can sometimes be distinctly seen during inspiration. On the other hand, when the diaphragm is pushed high and held there by the presence of fluid or a solid tumor, as in cirrhosis of the liver or leukæmia, the breathing has largely to be performed by the ribs, and becomes, as we say, costal in type (see below, p. 80).

2. *Anomalies of Expansion*

If we watch the patient while he takes a full breath, we may notice certain variations from the normal type of respiratory movements.

We may see: (1) Diminished expansion of one side (as a whole, or at the apex). (2) Increased expansion of the other side.

(1) If *diminished expansion* of one side is due to pleuritic effusion, pneumothorax, or solid tumor of the lung or pleura, the affected side is usually *distended* as well as *immobile*. When, on the other hand, the lung is retracted or bound down by adhesions, as in phthisis, old pleurisy, occlusion of the bronchus, or from the pressure of an aneurism, we have immobility combined with a *retraction* of the affected side. In tuberculous disease at the apex of the lungs we may see one side or both sides fail to expand at the top. Restriction of the motion of one side of the chest may also be due to pain or to pressure from below the diaphragm. An enlarged liver or spleen and tumors of the hepatic or splenic region may in this way prevent the normal expansion of one or the other side of the thorax. Occasionally a hemiplegia or a unilateral paralysis of the diaphragm results in diminished movement of one side of the chest.

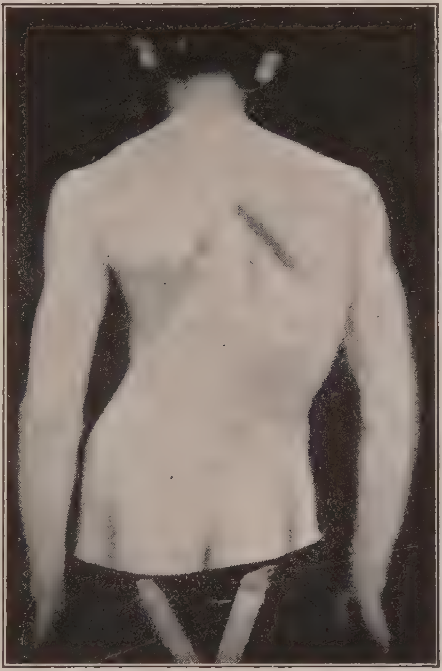

FIG. 70.—Lateral Curvature. Scoliosis.

(2) *Increased expansion* of one side of the chest is observed principally as a compensatory or vicarious overfunctioning of that side when the other side of the chest is thrown out of use by a large pleuritic effusion, by pneumothorax, long-standing pleurisy with contraction, or other causes.

3. *Dyspnœa*

This term is often used rather loosely to include: (1) Difficult breathing, whether rapid or slow. (2) Unusually deep breathing whether difficult or not. (3) Rapid breathing.

True dyspnœa or difficult breathing is almost always rapid as well, and does not differ at all from the well-known phenomenon of being "out of breath" after a hard run or any violent exertion. Conceive these conditions as persisting over hours or days, and we have the phenomenon known as dyspnœa. The breathing is not only quick but labored; that is, performed with difficulty, and unusual muscles, not ordinarily called upon for respiration, come into play and are seen working above the clavicle and elsewhere. More or less distress is generally expressed in the face, and there is often a blueness of the lips or a dusky color throughout the face. The commonest causes of dyspnœa are the various forms of heart disease, pneumonia, large pleuritic effusion, asthma, and phthisis. The researches of Barcoft and Thomas Lewis[1] make it probable that there are two types of dyspnœa—*Mechanical and Chemical.*

(*a*) *Mechanical dyspnœa* is associated with a decrease of O_2 and an increase of CO_2 in the blood, hence with a proportionate cyanosis. It is seldom marked in bed-ridden patients and does not increase at night unless the patient slips down from his sitting position. It is increased by pressing on the abdomen so as to drive blood to the heart. It is seldom if ever periodic.

(*b*) In *chemical dyspnœa* there is no excess of CO_2 in the blood and no considerable cyanosis. It is commonly seen in bed-ridden patients, is worse at night even when there is no orthopnœa and so no slipping down. It is not increased by abdominal pressure. Finally it is associated with the presence or increase of an unknown nonvolatile acid in the blood, in other words with acidosis. Thereby it is made comparable with the dyspnœic acidosis after long hard exercise (lactic acid from the contracting muscles) and with the acidosis of diabetic air hunger (β-oxybutyric acid). An acidosis has also been demonstrated in the hyperpnœa of normal persons at high altitudes, in diabetes, uræmia, lobar pneumonia, thyrotoxicosis, tuberculous pneumothorax and eclampsia.

This type of dyspnœa is often called "uræmic" but the term is too vague to be of value at present.

(*c*) The mechanical and chemical causes of dyspnœa often act simultaneously.

Dyspnœa may affect especially *inspiration*, as, for example, when a foreign body lodges in the larynx, or in ordinary "croup." In such cases we speak of "*inspiratory dyspnœa*," distinguishing it from "*expira-*

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[1] Thos. Lewis: Lectures on the Heart (P. B. Hoeber, New York, 1915).

tory dyspnœa" such as occurs in asthma and in barrel-chested persons. In the latter condition the breath seems to enter the chest readily, but the difficulty is to get it out again. Expiration is greatly prolonged and often wheezey.

Combined types in which both respiratory acts are difficult are more common.

Sucking-in of the interspaces in the lower axillary regions or below the clavicles may be seen in connection with dyspnœa whenever the lungs are prevented by some cause from properly expanding during inspiration. This may occur in obstruction at the glottis.[1]

Simple rapidity of breathing should be distinguished from dyspnœa. In adults the normal rate of respiration is about 18 per minute. In children, it is considerably quicker and more irregular. It is not desirable to attempt here to enumerate all the causes which may lead to a quickening of the respiration. Among the commoner are muscular exertion, emotion, hysteria, diseases of the heart and lungs, and fluid or solid accumulations below the diaphragm, which push up that muscle and cause it to encroach abnormally upon the thoracic cavity. Most of the infectious fevers are also apt to be accompanied by quickened breathing, especially but not exclusively when the fever is associated with a disease of the heart, lungs, pleura, or pericardium. Further chemical study of the blood will help us doubtless more accurately to divide and classify the varieties of breathlessness. The purely mechanical types are probably seen but rarely in bed-patients. In this, as in many other fields of medicine, the mechanical wanes and the chemical steadily waxes in etiological significance.

V. Relation of Dyspnœa to Cyanosis

These two phenomena go parallel in degree and coincide in appearance and disappearance so long as we are dealing with the mechanical type of obstruction to respiration (as in the rheumatic type of heart disease or in obstruction of the upper air passages). In the chemical or acid type of dyspnœa cyanosis is absent or disproportionately slight. On the other hand, in polycythæmia, in some extremely barrel-chested persons and in congenital heart disease, there may be striking cyanosis with little or no dyspnœa. Here it is the crowding of the peripheral capillaries with red blood cells that is chiefly responsible for the patient's color. An actual hyperactivity of the marrow exists in many of these cases and the red cells are increased not only at the periphery but in the larger blood vessels and internal organs ("primary or secondary polycythæmia").

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[1] Slight retraction of the lower interspaces in the axilla during inspiration is often seen in health. In disease this phenomenon is greatly exaggerated.

VI. CHANGES IN THE RESPIRATORY RHYTHM

1. *Asthmatic Breathing*

In asthma the normal rhythm is reversed; audible expiration becomes longer than inspiration. Inspiration may be represented by a short gasp, while expiration becomes a prolonged wheeze. Dyspnœa is usually very marked. In barrel-chested persons we get the same type of breathing so far as rhythm is concerned but the dyspnœa is not usually so extreme and the auxiliary muscles of respiration are not so apt to be called into use. In many barrel-chested persons one sees the thorax move all as one piece, "*en cuirasse*," owing to a senile fixation of the bones of the thorax from ossification of the cartilaginous portions. In hereditary syphilis or phthisis this fixation may occur in youth or early middle age. Weak heart and pulmonary œdema are usually the main factors in the so-called "emphysematous dyspnœa."

2. *Cheyne-Stokes Breathing*

An anomaly of respiratory rhythm in which short, recurrent paroxysms of dyspnœa are preceded and followed by periods in which no respiration occurs (apnœa). If we represent the normal respiratory movement by an up-and-down line, the Cheyne-Stokes type of breathing would appear as in Fig. 71. The period of apnœa may

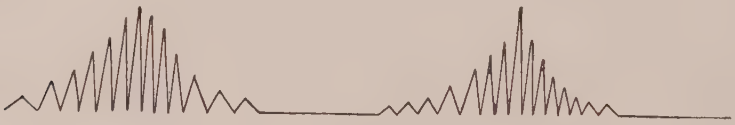

FIG. 71.—Cheyne-Stokes Respiration.

last from one to ten seconds; then short, shallow respirations begin and increase rapidly, both in volume and in rate, until a maximum of marked dyspnœa is reached, when a diminution in the rate and depth of the act begins, and the patient gradually returns to the apnœic state. The length of the whole paroxysm may be from 30 to 70 seconds. During the apnœic period the patient is apt to drop asleep for a few seconds and the pupils may become contracted. When the paroxysm of dyspnœa is at its height, he is apt to cough and shift his position restlessly, or in case the whole phenomenon occurs during sleep he moves uneasily in his sleep at this period. Modified types of this also occur, in which there is rhythmic increase and decrease in the depth and rapidity of respiration but without any intervening period of apnœa ("Biot's breathing"). This type of breathing is most often seen in various types of cardiac, renal, or cerebral disease. Conner has recorded many varieties in tuberculous meningitis. Respiratory arrhythmias are generally more marked at night but may occur only at that time. Often in healthy children and occasionally in sound adults, Cheyne-Stokes breathing occurs during sleep and rarely even in waking hours. It is especially apt to occur when the person lies on the back and may be checked either in health or disease by turning on the side (Edsall). As a rule continuous Cheyne-Stokes breathing is a sign of grave prognostic significance, but patients have been known to recover completely after weeks or even months of it. It is due probably to a relative numbness of the respiratory center which allows its usual chemical excitants to accumulate (in the apnœic pause) until they can break through its insensibility and make it start the breathing again.

3. *Restrained or "Catchy" Breathing*

When the patient has a "stitch in the side," due to dry pleurisy, or to other causes, the inspiration may be suddenly interrupted in the middle, owing to a seizure of pain which makes the patient stop breathing as quickly as he can. The same conditions may produce very shallow breathing as the patient tries to avoid the pain which a full inspiration will cause. This type of restrained breathing is often seen in pleurisy and pneumonia, and in the latter disease expiration is often accompanied by a little moan or grunt of discomfort.

Shallow and irregular breathing is often seen in states of profound unconsciousness from any cause, sucn as apoplexy or poisoning. A few deep respirations may be followed by a number of shallow and irregular ones.

Sterno-mastoid breathing. When death is imminent in any disease, the respiration may become very irregular and gasping, and it is apt to be accompanied by a peculiar nodding movement of the head, the chin being thrown quickly upward during inspiration and falling slowly during expiration. I have known but one patient to recover after this type of breathing had set in.

After severe hemorrhage the breathing may be of a *sighing type* as well as very shallow.

4. *Stridulous Breathing*

A high-pitched, crowing or barking sound is heard during inspiration when there is obstruction of the entrance of air at or near the glottis. This type of breathing occurs in spasm or œdema of the glottis, "croup," laryngismus stridulus and post-pharyngeal abscess; it forms the "whoop" in the paroxysms of whooping-cough. Laryngeal or tracheal obstructions due to foreign bodies, to tumors within or pressure from without the air-tubes, may cause a similar type of respiration. It is in these cases more especially that we see the sucking-in of the interspaces mentioned above (see p. 75).

VII. DIAPHRAGMATIC MOVEMENTS

1. *The Phrenic Wave*

The normal movements of the diaphragm may be rendered visible by the following procedure, suggested by Litten in 1892: The patient lies upon his back with the chest bared and the feet pointed directly toward a window. Cross lights must be altogether excluded by darkening any other windows which the room may contain[1] (see Fig. 72). The observer stands at the patient's side. As the ribs rise with the

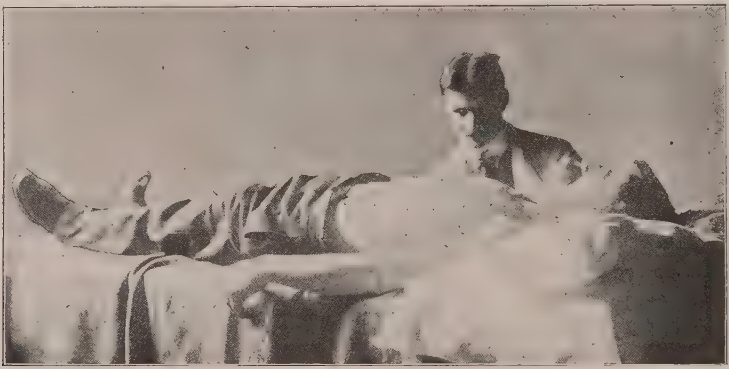

FIG. 72.—Litten's Diaphragm Shadow. Proper position of patient and of observer. The shadow is best seen near L.

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[1] If it is convenient to move the patient's bed into the proper position with relation to the window, or if the foot-board interferes, or if the observation has to be made after dark, a dark lantern or electric hand lantern held in the hand answers very well.

movement of inspiration, a short, narrow shadow moves down the axilla from about the seventh to about the ninth or tenth rib. During expiration the shadow rises again to the point from which it started, but is less easily seen. This phenomenon is to be seen on both sides of the chest, less well in the back, and sometimes in the epigastrium. It is best seen in spare, muscular young men, but is never absent in health except in those who are very fat, or who cannot or will not breathe deeply. The latter condition occurs in hysteria and in some very stupid persons. In the observation of several thousand cases, I have never known it absent in health except under these conditions.

In normal chests, the excursion of the shadow is about two and a half inches; with very forced breathing three and a half inches. The mechanism of this phenomenon is best understood by imagining a coronal section of the thorax as seen from the front or back (see Fig. 73). At the end of expiration, the diaphragm lies flat against the thorax from its attachment up to about the sixth rib. During inspiration it "*peels off*" as it descends and allows the edge of the lung to come down into the chink between the diaphragm and thorax. This "peeling off" of the diaphragm and the descent of the lung during inspiration give rise to the moving shadow above described.

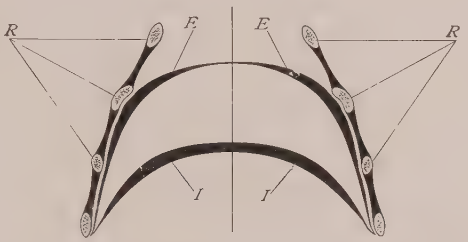

FIG. 73.—Excursion of the Diaphragm during Forced Respiration. *R*, Ribs; *E*, position of the diaphragm at end of expiration; *I*, position of diaphragm at end of inspiration.

By thus observing the excursion of the diaphragm we can obtain a good deal of information of clinical value.

In pneumonia of the lower lobe, pleuritic effusion, extensive pleuritic adhesions, or in well marked barrel chest, the shadow is absent. This is explained by the fact that in these conditions the diaphragm is held off from the chest wall so that its movements communicate no shadow. In pleuritic adhesions the movements of the diaphragm are prevented. In early phthisis I have generally found the excursion of the diaphragm diminished upon the affected side, owing to a loss of elasticity in the effected lung and in part probably to pleuritic adhesions. On the other hand, fluid or solid tumors below the diaphragm, unless very large, do not prevent the descent of that muscle, and so do not abolish the diaphragm shadow. In cases in which the diagnosis is in doubt between fluid in the right pleural cavity and an enlargement of the liver upward or a subdiaphragmatic abscess, the preservation of the Litten's phenomenon in the latter two affections may be of some value in diagnosis. Very large accumulations of ascitic fluid may so far restrain the diaphragmatic movements that no shadow can be seen.

This method frees us to a considerable extent from the need of using the spirometer to determine the capacity of the lungs. By measuring the excussion of the phrenic shadow and taking account of the thoracic movement, we obtain a fair idea of the respiratory capacity (so called "vital capacity") of the individual.

2. *The Ill Effects of a High Diaphragm*

(*a*) The permanently raised position into which the midriff is forced by abdominal stasis with ascites and engorged liver, by extreme obesity, by abdominal tumors or dropsies and by gaseous distention, is an important factor in the dyspnœa and in the pulmonary lesions which these conditions produce. Pulmonary engorgement, infarct and broncho pneumonia are favored by high diaphragm.

(*b*) Moreover since the pumping action exerted by the midriff on the blood of the splanchnic area is an important factor in maintaining the circulation, the feeble, restricted motions of a high diaphragm handicap the heart and throw extra work upon it.

(*c*) Lewis has shown that free diaphragmatic breathing raises blood pressure and that a purely or largely costal breathing lowers it. Lowered blood pressure appears therefore to be a further disadvantage resulting from a high fixed position of the diaphragm.

Permanently low diaphragm (splanchnoptosis) is believed to produce circulatory disturbance since in its steady depression (as in its permanent exaltation) it cannot pump along the splanchnic blood effectively.

Unilateral displacement of the diaphragm will be further referred to in the section on subphrenic abscess.

VIII. Observation of the Cardiac Movements

1. *The Normal Cardiac Impulse*

With each systole of the heart there may be seen in the great majority of normal chests an outward movement of a small portion of the chest wall just inside and below the left nipple. This phenomenon is known as the cardiac impulse.[1] It is now generally admitted that the "apex impulse" is caused by the impact of a portion of the right ventricle against the chest wall and not by the apex of the heart itself. (The bearings of this fact, which have not, I think, been generally appreciated, will be discussed presently.) The position of the *maximum* impulse in adults is usually in the fifth intercostal space just inside the nipple line. In children under the age of six it is often in the fourth interspace and outside the nipple; while in persons with low diaphragm it may descend as low as the sixth interspace. In adults it is occasionally absent even in perfect health and under certain pathological conditions to be later mentioned.

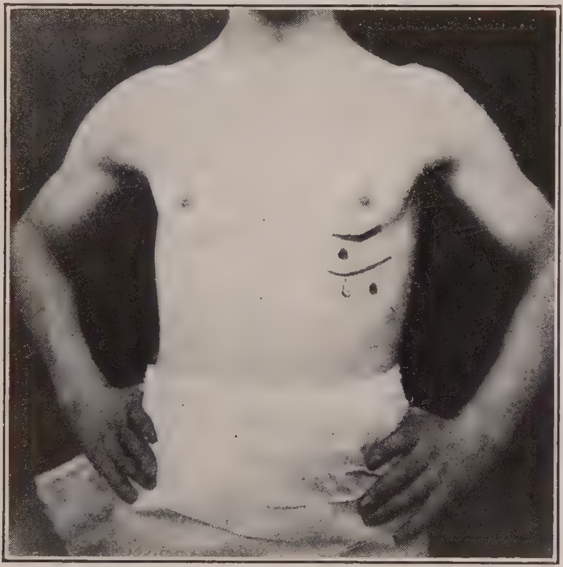

FIG. 74.—Showing Amount of Shifting of the Apex Impulse with Change of Position. The inner dot represents the position of the impulse when the patient lies on his back; the outer dot corresponds to the position of the apex with patient on left side.

(*a*) The position of the impulse varies to a certain extent according to the position of the body. If the patient lies upon the left side, the heart's apex swings out toward the axilla, so that the visible impulse shifts from one to two and one-half inches to the left (see Fig. 74).

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[1] For a more detailed description of the normal position of the cardiac impulse, see next page.

A slight shift to the right can also be brought about by lying upon the right side, and, as a rule, the impulse is less visible in the recumbent than in the upright position. Since the heart is lifted with each expiration by the rise of the diaphragm and falls during inspiration, a corresponding change can be observed in the apex beat, which, in forced breathing, may shift as much as one interspace. Of the changes in the position of the impulse brought about by disease, I shall speak in a later paragraph.

(*b*) *Relation of the maximum cardiac impulse to the apex of the heart.*—I mentioned above that the maximum cardiac impulse is not due to the striking of the apex of the heart against the chest wall, but to the impact of a portion of the right ventricle. The practical importance of this fact is this: When we are trying to localize the apex of the heart in order to determine how far the organ extends to the left and downward, it will not do to be guided by the position of the *maximum* impulse, for the apex of the heart is almost always to be found three-fourths of an inch or more farther to the left (see Fig. 75). This may be proved by percussion (*vide infra*, p. 125), and by fluoroscopy. The true position of the cardiac apex thus determined corre-

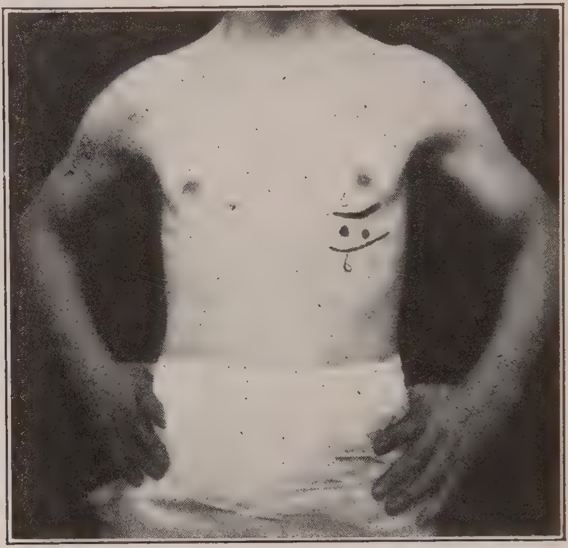

FIG. 75.—The Inner Dot is the Maximum Cardiac Impulse. That to the right is the true apex of the heart, as obtained by percussion. The ribs are numbered.

sponds usually not with the *maximum* impulse, but with the point farthest out and farthest down at which *any localized rise and fall* synchronous with the heart beat *can be felt*.

(*c*) Besides the definite and localized impulse which has just been described, it is often possible to see that a considerable section of the chest wall in the precordial region is lifted "*en masse*." The phenomenon is the "*Herzenstoss*" of the Germans, with which the "*Spitzentoss*" or *apex impluse* is contrasted. A variable amount of "*Herzentoss*" can be seen and felt over any normal heart when it is acting rapidly and forcibly, and in thin, nervous subjects or in children even when the heart is beating quietly. It is more marked in cardiac neuroses, in Graves' disease, when the heart is hypertrophied, and when there is more or less stiffening of the ribs with loss of their natural elasticity. At times it may be impossible to localize any one point to which we can give the name of apex impulse, and what we see is the rhythmical rise and fall of a section of the chest as large as the palm of the hand or larger. In many fat people and in those with thick, stiff chest walls it is often impossible to make out any cardiac impulse. Sometimes we can feel it when the patient lies on his left side, and allowing for its normal lateral swing, we may in this way calculate its ordinary position. In old people palpation is often useless and we fall back on percussion, blood pressure and the *x*-ray.

(*d*) *X-ray measurements of the normal heart.*—The normal adult heart *x*-rayed in the upright position at a distance of 7 feet extends from 7 to 11 cm. to the left of the median line and from 3 to 5 cm. to the right of the median line. The heavier the person the larger the heart. Women's hearts average about 1 cm. smaller in each direction.

The transverse diameter of the great vessels is usually between 4 and 6.5 mm.

Percussion agrees fairly well with the measurements to the left of the median line (where also palpation usually serves us excellently) but is unreliable on measurements to the right of the median line.

(*e*) *Character of the cardiac impulse.*—Palpation is considerably more effective than inspection in giving us information as to the nature of the cardiac movements which give rise to the "apex beat," but even inspection sometimes suffices to show that the impulse has the *slow* forcible thrust characteristic of hypertrophy or is of the nature of a short tap, a peristaltic wave, or a diffuse slap against the chest wall. In some cases a distinct undulation can be seen passing from the apex region upward toward the base of the heart, or in the opposite direction.

2. *Displacement of the Cardiac Impulse*

To one familiar with the position, extent, and character of the normal cardiac impulse, any displacement of this impulse from its normal site or any superadded pulsation in another part of the chest is apparent at a glance. I will consider first the commonest forms of dislocation of the apex impulse.

(*a*) *Displacement of the cardiac impulse due to hypertrophy and dilatation of the heart.*—By far the most common directions of displacement are toward the left axilla and downward. As a rule, it is displaced in both these directions at once. I shall return to this subject more in detail under the heading Cardiac Hypertrophy, but here I may say that enlargements of the left ventricle tend especially to displace the apex impulse downward, while enlargements of the right ventricle are more commonly associated with displacement of the impulse toward the axilla.

(*b*) Next to hypertrophy and dilatation of the heart perhaps the commonest cause of dislocation of the cardiac impulse is pressure from below the diaphragm. When the diaphragm is raised by a large accumulation of gas or fluid, by pregnancy or by solid tumors of large size, we may see the apex beat of the normal heart in the fourth interspace and often an inch or more inside the nipple line.

(*c*) Of nearly equal frequency is displacement of the heart due to left-sided *pleuritic effusion* or to pneumothorax (see below, p. 322).

When a considerable amount of air or fluid accumulates in the left pleural cavity, the heart is displaced bodily to the right so that it may be concealed behind the sternum or be visible beyond it to the right; in extreme cases it may be dislocated beyond the right nipple. Right pleuritic effusions have far less effect upon the position of the cardiac impulse, but when a very large amount of fluid accumulates we may see the impulse displaced considerably toward the left axilla.

(*d*) I have mentioned causes tending to push the heart to the *right, to the left, or upward.* Occasionally the heart is pushed *downward* by an aneurismal tumor or a neoplasm of the mediastinum. In these cases there is usually more or less displacement to the left as well. As a result of arteriosclerosis or cardiac hypertrophy the aorta may sag or stretch a little, the diaphragm may stand lower, so that the apex beat descends to the sixth interspace, or (more often) is lost to sight and touch *behind the bunch of convergent costal cartilages* just to the left of the ensiform. Very frequently in barrel-chested men past forty-five the whole heart sinks considerably, so that a marked systolic retraction (less often pulsation) is seen below the ensiform in the epigastrium.

(*e*) Displacement of the cardiac impulse resulting from *adhesions* of the pericardium to the pleura, with subsequent contraction, occurs in *fibroid phthisis* and in some cases of long-standing disease of the pleura. Through the effect of negative pressure the heart may be sucked into the space formerly occupied by a portion of the lung, when the latter has become contracted by disease. It seems probable, however, that in the majority of cases adhesions between the pleura and pericardium play a part in such displacement. By these means the heart may be displaced to the right of the sternum, as it is by left-sided pleuritic effusion. It is often drawn upward as well as to the right in such cases because the contraction takes place in the upper part of the lung. More rarely we see the heart drawn toward the left clavicle in fibroid phthisis of the left apex.

(*f*) Distortion of the thorax due to spinal curvature or other causes may bring about a considerable displacement of the heart from its normal position.

(*g*) *Dextrocardia and Situs Inversus.*—In rare cases a displacement of the apex impulse to the right of the sternum may be due either to a *transposition of all viscera* (the liver being found upon the left, the spleen upon the right, etc.), or to *dextrocardia*, in which the heart alone is transposed while the other viscera retain their normal places.

(*a*) *Summary*

The apex impulse is displaced by:

(*a*) Hypertrophy and dilatation of the heart.
(*b*) Pressure from below the diaphragm.
(*c*) Air or fluid in one pleural cavity especially the left.
(*d*) Aneurism, mediastinal growths, and sagging of the aorta.
(*e*) Fibroid phthisis and chronic pleurisy.
(*f*) Spinal curvature.
(*g*) Transposition of the heart or of all the viscera.

3. *Apex Retraction*

Before leaving the subject of the cardiac impulse it seems best to speak of those cases in which during systole we see a *retraction* of one or more interspaces at or near the point where the cardiac impulse normally appears.

(*a*) In by far the greater number of instances such retraction is due to negative pressure produced within the chest by the vigorous contraction of a more or less hypertrophied and dilated heart. In these cases the retraction is usually balanced by an impulse in the next interspace so that a "walking beam" appearance or tilting of a piece of the chest wall results.

(*b*) In rarer cases several interspaces, both in the precordial region and in the left lower axilla and back, may be drawn in as a result of adhesions between the pericardium and the chest wall, such as form in cases of adherent pericardium and fibrous mediastinitis[1] (see below, pages 245 and 246).

4. *Epigastric Pulsation*

In a considerable portion of healthy adults a pulsation or retraction at the epigastrium synchronous with the systole of the heart is to be seen from time to time. Such pulsation has often been considered evidence of hypertrophy of the right ventricle, but atopsy findings do not substantiate this belief. In some cases epigastric pulsation is to be explained as the transmission of the heart's impulse through the liver, or as a lifting of that organ by the movements of the abdominal aorta. In other cases it is due to bathycardia ("low heart"—a condition very common in arteriosclerosis).

5. *Visible Pulsations due to Uncovering of Portions of the Heart Normally Covered by the Lungs*

One of the commonest causes of visible pulsations in parts of the chest where normally none is to be seen is *retraction of the lung*.

(*a*) In chlorosis we saw such pulsations frequently before that disease became so rare. In that disease, as in other debilitated states, the lungs are often not adequately expanded owing to the superficiality of the respiration, and accordingly their margins do not cover as much of the surface of the heart as they do in healthy adults. This results in rendering visible, in the second, third, or fourth left interspace near the sternum, pulsations transmitted from the conus arteriosus or from the right ventricle. Less commonly, similar pulsations due to the uncovered aorta may be seen on the right side of the sternum.

(*b*) A rarer cause of retraction of the lungs is phthisis or chronic interstitial pneumonia. In these diseases a very large area of pulsation may be seen in the precordial region owing to the entire uncovering of the heart by the retracted lung, even when the heart is not drawn out of its normal position.

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[1] Or more often as a result of simple cardiac hypertrophy. Most cases of aortic regurgitation in thin young patients show this systolic retraction in the left lower back.

IX. Aneurism and Other Causes of Abnormal Thoracic Pulsation

So far I have spoken altogether of pulsations transmitted directly to the thorax by the heart itself, but we have also to bear in mind that an aneurism may transmit to the chest wall pulsations which it is exceedingly important for us to recognize and properly to interpret.

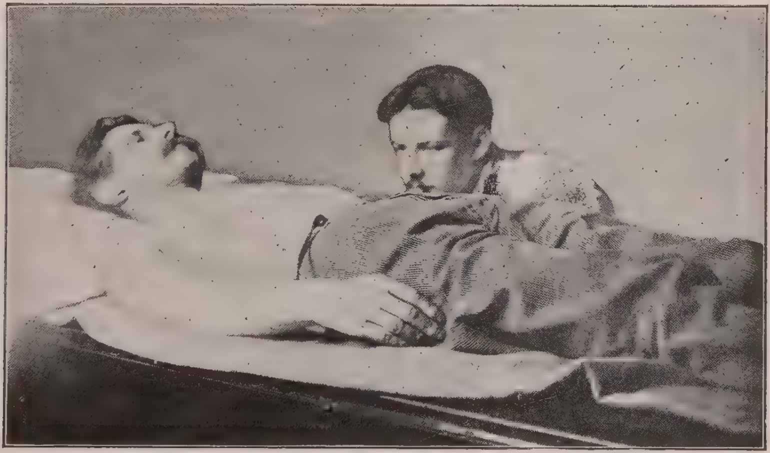

Fig. 76.—Position When Looking for Slight Aneurismal Pulsation.

No disease is easier to recognize than aneurism when the growth has perforated the chest wall and appears as a tumor externally, but it is much more important as well as much more difficult to recognize the disease while it is confined within the thorax. In such cases, the movements transmitted from the aorta to the chest wall may be so slight that only the keenest and most thorough inspection controlled by palpation will detect them. When slight pulsations are searched for, the patient should be put in the position shown in Fig. 76, and the observer should place himself so that his eye is as nearly as possible on a level with the chest and looks across it so that he sees it in profile. In this position, or in a sitting position with tangential light, he can make out pulsations which are totally invisible if the patient sits facing the light.

Pulsations due to aneurism are most apt to be seen in the first or second right interspace near the sternum, and not infrequently the clavicle and the adjacent parts may be seen to rise slightly with every beat of the heart, but in any part of the chest wall pulsations due to an aneurism are occasionally to be seen, and should be looked for scrupulously whenever the symptoms of the case suggest the possibility of this disease (see below, p. 256).

1. *Pulsating Pleurisy*

In cases of purulent pleurisy in which the fluid has worked its way out between the ribs so that it is covered only by the skin and subcutaneous tissues, a pulsation transmitted from the heart may become visible, and the resemblance to the pulsation seen in aneurism may be confusing. Such pulsation is apt to be seen in the upper and front portions of the chest. Very rarely a pleuritic effusion which has not burrowed into the chest wall may transmit to the latter a wavy movement corresponding to the motions set up in the fluid by the cardiac contractions. I have never seen a pulsating pleurisy.

X. Inspection of the Peripheral Vessels

In the study of all diseases of the heart and lungs it is important to take account of all vascular phenomena apparent in the neck or in the extremities, since such phenomena have a very direct bearing upon the interpretation of the conditions obtaining within the chest. Inspection plays a very large part in the study of these vascular phenomena. We should look for: (*a*) Venous phenomenia; (*b*) Arterial phenomena; (*c*) Capillary phenomena.

1. *Inspection of the Veins*

1. The condition of the veins of the neck is of considerable importance in the diagnosis of diseases of the heart and lungs. Where the tissues of the neck are more or less wasted, the veins may be quite prominent even when no disease exists within the chest, and in such cases they may be more or less distended furing each expiration, especially if dyspnœa or cough is present. If the overdistended veins are completely emptied during deep inspiration and on both sides of the neck, we can usually infer that there is an overdistention of the right side of the heart. When a similar phenomenon occurs on one side only, it may mean pressure upon one innominate vein. So far I have spoken of venous changes synchronous with respiration, but we may have also

2. *A presystolic pulsation or undulation* seen either in the external jugular vein or in the bulbus jugularis between the two attachments of the sternomastoid muscle. Such pulsation or undulation, which is to be seen just before each systole of the heart, is not necessarily anything abnormal and must be carefully distinguished from

3. *Systolic venous pulsation*, such as occurs in what I suppose is one of the most common valvular diseases of the heart, relative tricuspid regurgitation,[1] as well as in a good many other conditions.

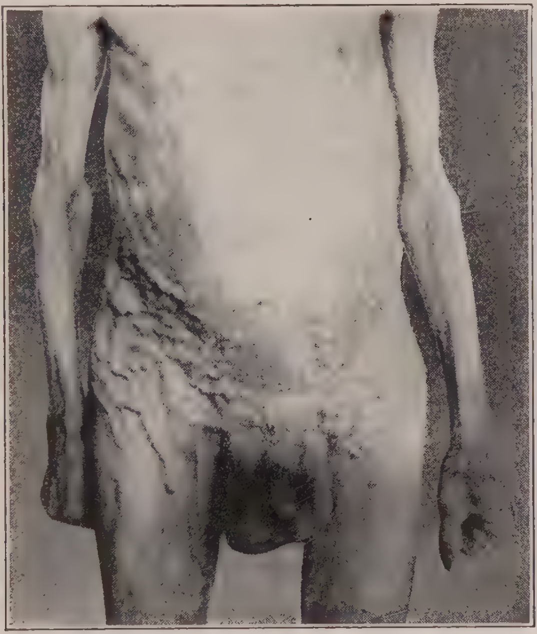

FIG. 77.—Tortuous Veins on Chest and Abdomen. (Autopsy showed obliteration of the vena cava inferior.)

Systolic venous pulsation is more often seen upon the right side than upon the left side of the neck. There may be a wave during the systole of the auricle and another during the systole of the ventricle, the latter closely following the former. In any case in which a doubt arises whether a pulsation in the veins of the neck is due to tricuspid regurgitation, it is well to try the experiment of emptying the vein by stroking it from below upward. If it immediately fills from below, we may be practically certain that tricuspid regurgitation is present. In the vast majority of cases of venous pulsation due to other causes or occurring in healthy persons a vein will not refill from below if emptied in the manner above described. The finer points relating to the variations in the cervical venous pressure are recognized by phlebograms traced by MacKenzie's instrument or in some similar way.

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[1] A pulsating carotid may transmit an up-and-down motion to the veins overlying it. In such cases, if the veins be emptied by "milking" them upward, they will not refill from below.

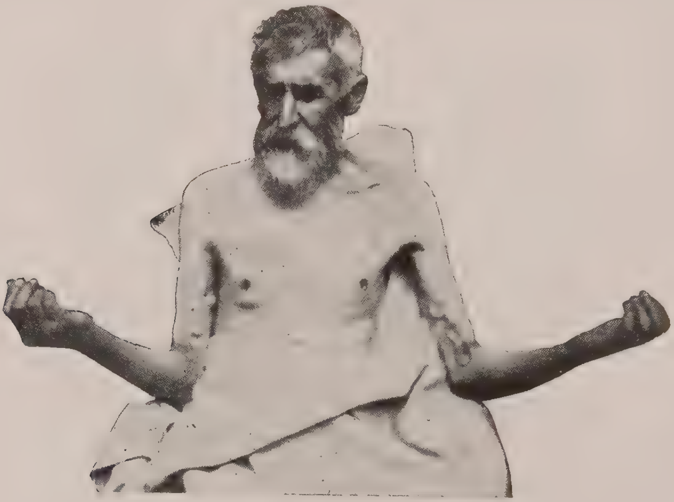

FIG. 78.—Enlarged and Tortuous Brachial Arteries (Arterio-sclerosis).

The venous waves so recorded tell us much that is interesting and some things that are important about the behavior of the auricles. (See below, p. 117.)

4. Rarely, superficial veins may be seen to pulsate in other parts of the body, especially in aortic regurgitation, and occasionally large and tortuous veins may be seen pulsating upon the thoracic or abdominal wall, representing an attempt at collateral circulation when one or the other vena cava is compressed (Fig. 77).[1]

2. *Arterial Phenomena*

1. In thin or nervous persons pulsations are not infrequently to be seen in the carotids independent of any abnormal condition of the heart.

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[1] Enlarged veins about the navel, the so-called "caput Medusæ," are commonly found in text-books, but rarely in cirrhosis of the liver. I have never seen them.

2. Very violent throbbing of the carotids, more noticeable than the normal, often occurs in severe anæmias and occasionally in simple overaction of the heart, *e.g.*, in Graves' disease. From the same causes, visible pulsation may occur in the subclavian, axillary, brachial, and radial arteries, as well as in the large arterial trunks of the lower extremity.

3. In arterio-sclerosis occurring in spare, elderly men, one often notices a lateral excursion of the tortuous brachial arteries synchronous with every heart beat. An up-and-down pulsation may occur at the same time. Not infrequently the arteries which are stiffened by arterio-sclerosis (see below, page 108) stand out visibly as enlarged, tortuous cords along the inner side of the biceps muscle (see Figs. 78 and 79), and occasionally the course of the radial artery may be traced over a considerable distance in the forearm. In rare cases inequalities produced in the arterial wall by deposition of lime salts may be visible as well as palpable. The temporal artery (unlike the brachial) is *normally* tortuous. Its course has therefore no relation to arterio-sclerosis. It is visible in many normal persons.

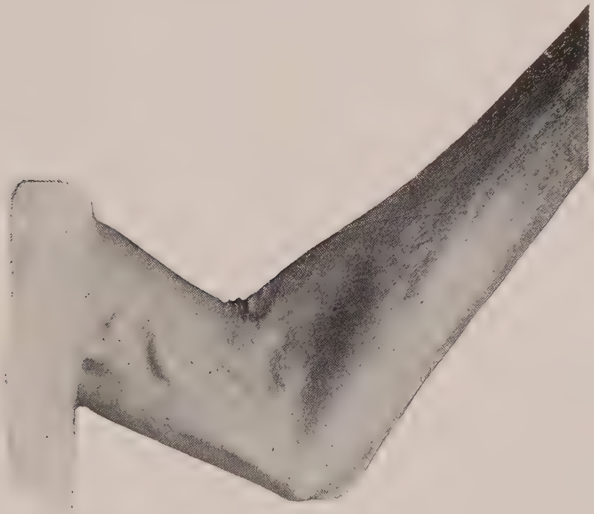

FIG. 79.—Enlarged and Tortuous Brachial Artery (Arterio-sclerosis).

3. *Capillary Pulsation*

If a microscopic slide is placed against the mucous membrane of the lower lip so as partially to blanch its surface, one may see, with each beat of the heart (in cases of aortic regurgitation and in some other conditions presently to be mentioned), a delicate flushing of the blanched surface beneath the glass slide. The same pulsation is sometimes to be observed under the finger nails, or may be still better brought out by holding a pocket electric flash light against the finger pulp so as to transilluminate it. The paling and flushing with each heart beat is well seen. This phenomenon will be referred to again when we come to speak of aortic regurgitation. Here it suffices to say that it is not in any way peculiar to that disease, but occurs occasionally in health, in anæmia, in thyrotoxicosis, *i.e.* in conditions associated with large pulse pressures (see below, pp. 112–114), as well as in any area of inflammatory hyperæmia (jumping toothache, throbbing felon, etc.).

XI. Inspection of the Skin and Mucous Membranes

Light may be thrown upon the diagnosis of very many diseases by observing the color and condition of the cutaneous surfaces as well as of the mucous membranes. We should look for the following conditions:

(1) Cyanosis.
(2) Œdema.
(3) Pallor.
(4) Jaundice.
(5) Scars and eruptions.

1. *Cyanosis*

By cyanosis we mean a purplish or grayish blue tint noticeable especially in the face, in the lips, and under the nails. There are many degrees of cyanosis, from the slight purplish tinge of the lips, which a little overexertion or slight exposure to cold may bring out, up to the gray-blue color seen in advanced cases of pulmonary or cardiac disease, or the dark reddish-blue seen in congenital malformations of the heart. Cyanosis makes a very different impression upon us when it is combined with pallor on the one hand or with jaundice on the other. When combined with pallor, one gets various ashy-gray tints, while the admixture of cyanosis and jaundice results in a color very difficult to describe, sometimes approaching a greenish hue. The commonest causes of cyanosis are:

(*a*) Valvular or parietal disease of the heart.
(*b*) Extreme types of the barrel chest.

(*c*) Asthma.

(*d*) Pneumonia.

(*e*) Phthisis.

(*f*) Obstruction of the superior vena cava by mediastinal tumors or other causes.

(*g*) In some persons a certain degree of cyanosis of the lips exists despite perfect health. This is especially true of weather-beaten faces and those of the so-called "full-blooded" type.

(*h*) Methæmoglobinæmia, such as occurs after the excessive use of coal-tar analgesics (antifebrine in headache powders, etc.).

A rare but very striking type of cyanosis is that seen in cases of congenital heart disease, in which the lips may be indigo blue in color or almost black while yet no dyspnœa is present.

Cyanosis of intestinal origin has been described by English writers. It is distinctly rare.

In polycythæmia the face and lips may show the ordinary tint of cyanosis or may be of a deep red peculiar to this disease.

2. *Œdema*

Œdema, or the accumulation of serous fluid in the subcutaneous spaces, is usually appreciated by palpation rather than by inspection, but sometimes makes the face look very puffy, especially under the eyes. This is not a common occurrence in diseases of the chest, in connection with which such œdema as takes place is usually to be found in the lower extremities and is appreciable rather by palpation than by inspection. If we are not familiar with a patient's face, we often do not perceive in it the changes of outline due to œdema which a friend would notice at once. Clothing is apt to leave grooves and marks wherever it presses tightly upon the œdematous tissues, as around the waist or over the shoulders. In the legs, the presence of œdema may be suggested by an unnaturally smooth, glossy appearance of the skin. Such impressions, however, may be false unless controlled by palpation, for simple obesity may produce very similar appearances.

3. *Pallor*

Pallor suggests, though it does not in any way prove, anæmia. Pallor of the mucous membranes, as seen in the lips and conjunctivæ, is much more apt to be a sign of real anæmia than is pallor of the skin. At best, pallor is only a sign which suggests to us to look further into the case in one or another direction, and of itself proves nothing of importance. Hæmorrhage, cancer, nephritis, septicæmia and pernicious anæmia are the commonest causes of anæmia. *Pallor without anœmia* is often seen in tuberculosis, in arteriosclerosis and in the psychoneuroses.

4. *Jaundice*

The yellowish tint which appears in the skin, and especially in the conjunctivæ, when the escape of bile from the liver is hindered (catarrhal jaundice, gall-stones, cancer, cirrhosis), or when rapid hemolysis has occurred (malaria, sepsis).

5. *Scars and Eruptions*

The scars of old tuberculous glands in the neck, the scars of varicose ulcers along the shin bones, the various scars and eruptions of syphilis, of the exanthemata, of traumata and of surgical operations are of value in tracing the past history and interpreting the present illness. Without attempting to enter the field of dermatology it may be here mentioned that for the internist the skin lesions most important of recognition are those just mentioned and, in addition, drug eruptions, cutaneous neoplasms and the various causes of pigmentation.

XII. Enlarged Glands

Routine inspection may reveal the presence of enlarged glands in the neck or axillæ or groins, and may thereby give us a clew to the nature of the underlying disease; for example, the presence of enlarged glands in the neck, especially if there are any scars, sinuses, or other evidence that suppuration is going on or has formerly taken place in them, suggests the possibility of glandular tuberculosis or of an enlargement of the bronchial and mediastinal glands. In children cervical adenitis is most often a sign of bad teeth, tonsillitis and head lice. Again, malignant disease of the chest or abdomen is sometimes associated with the metastatic nodules over the clavicle (see Fig. 80), and a microscopic examination of them may thus reveal the nature of the intrathoracic disease to which they are secondary. Very large and matted masses of glands above the clavicle, which have never suppurated and have been painless and slow in their growth, suggest the presence of similar deposits in the mediastinum as a part of the symptom complex known as "Hodgkin's disease." The presence of a goitre or enlargement of the thyroid gland may account for a well-marked dyspnœa. Axillary adenitis means most often peripheral sepsis, next tuberculosis, then metastatic cancer, leucæmia and Hodgkin's disease. Inguinal adenitis (suppurative) is most often a result of gonorrhœa. If non-suppurative, it is usually due to sepsis in the leg, syphilis, leucæmia, Hodgkin's disease and metastatic cancer.

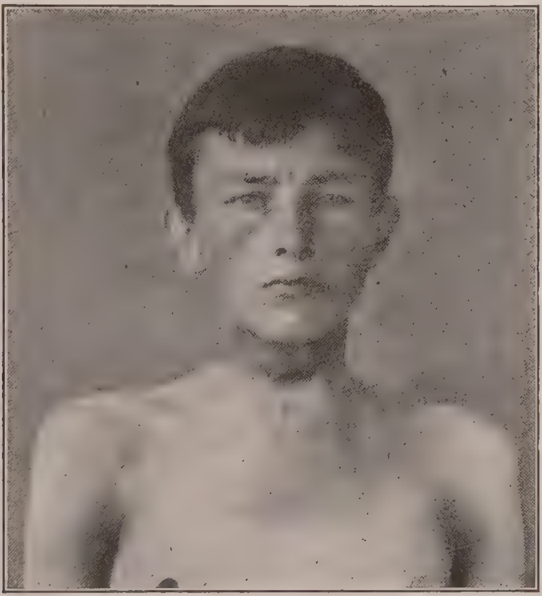

FIG. 80.—Sarcoma of Sternum and Cervical Glands. (Curschmann.)

Syphilis produces general glandular enlargement; the posterior cervical and the epitrochlear glands are often involved, but this is also the case in many diseases other than syphilis.

CHAPTER V

PALPATION AND THE STUDY OF THE PULSE

I. Palpation

The most important points to be determined by palpation—that is, by laying the hand upon the surface of the chest—are:

(1) The position and character of the *apex beat* of the heart.

(2) The presence of a "*thrill*" (see below).

(3) The vibrations of the spoken voice ("*tactile fremitus*").

(4) The presence of pleuritic or pricardial *friction.*

(5) Local muscular spasm.

Other less important data furnished by palpation will be mentioned later.

1. *The Apex Beat*

(*a*) In feeling for the apex impulse of the heart, one should first lay the palm of the hand lightly upon the chest just below the left nipple. In this way, we can appreciate a good deal about the movements of the heart, and confirm or modify what we have learned by inspection. One learns, in the first place, whether the heart beat is *regular* or not, and in case it is irregular, something as to the nature and degree of the arrhythmia; further one gets a more accurate idea than can be obtained through inspection regarding the *character of the cardiac movements*. The powerful, slow, widespread impulse of a hypertrophied heart, the sudden rap characteristic of mitral stenosis may be thus appreciated.

(*b*) After this, it is best to lay the tips of two or three fingers over the point where the maximum impulse is to be seen, and follow it outward and downward until one arrives at the point farthest to the left and farthest down at which it is still possible to feel any up-and-down movement. The point usually corresponds with the apex of the heart, as determined by percussion or fluoroscopy. *It does not correspond with the maximum cardiac impulse*, but is often to be found at least an inch farther to the left and downward (see above, Fig. 80).

Sometimes one can localize by palpation a cardiac impulse which is not visible; on the other hand, in some cases we can see pulsations that we cannot feel. Both methods must be used in every case.

When x-ray measurements cannot be had, palpation and inspection of the region give us the most reliable data that we have regarding the size of the heart. Percussion is often interfered with by the presence of gas in the stomach, of fluid or adhesions in the pleural cavity, or by the ineptness of the observer, but it is almost always possible with a little care to make out by a combination of palpation and inspection the position of the apex of the heart. When we can neither feel it nor see it, we may have to fall back upon auscultation, considering the apex of the heart to be at or near the point at which the heart sounds are heard loudest. When endeavoring to find the apex of the heart, we must not forget that the position of the patient influences considerably the relation of the heart to the chest walls. If the patient is leaning toward the left or lying on the left side, the apex will swing out several centimeters toward the left axilla. If the peripheral blood pressure is permanently high, it is well to conclude that the heart is enlarged, whatever the other physical signs.

2. *Thrills*

When feeling for the cardiac impulse with the palm of the hand, we are in a good position to notice the presence or absence of a very important physical sign to which we give the name of "thrill." The feeling imparted to the fingers by the throat of a purring cat is very much like the palpable "thrill" over the precordia in certain disease of the heart to be mentioned later. It is a vibration of the chest wall, usually confined to a small area in the region of the apex impulse, but sometimes felt in the second right intercostal space or elsewhere in the precordial region. This vibration or thrill almost always occurs intermittently, *i.e.*, only during a portion of the cardiac cycle. When felt in the apex region, it usually occurs just before the cardiac impulse; this fact we express by calling it a "*presystolic thrill;*" but occasionally we may feel a *systolic* thrill at the apex—one, that is, which accompanies the cardiac impulse. The word *thrill* should be used to denote only a purring, vibrating sensation communicated to the fingers by the chest wall. It is incorrect to speak of a thrill as if it were something audible.

We must especially distinguish a thrill from the slight systolic shudder or jarring which is often palpable over the whole precordia in functional neuroses of the heart or in conditions of mental excitement.

As a rule we can appreciate a thrill more easily if we lay the palm very lightly upon the chest, using a little pressure as possible. Firm pressure may prevent the occurrence of the vibrations which we desire to investigate. Of the thrills felt over the base of the heart, more will be said in Chapter X. At the apex nothing should be called a "thrill" unless it lasts longer than a normal heart sound. Many a forcible first sound in an excited, nervous subject is miscalled a thrill.

3. *Vibrations Communicated to the Chest Wall by the Voice*

"*Tactile fremitus*" is the name given to the sense of vibration communicated to the hand if the latter is laid upon the chest while the patient repeats some short phrase of words. The classical method of testing tactile fremitus is to ask the patient to count "one, two, three," or to repeat the words "ninety-nine" while the palm or the edge of the hand is laid upon the chest. The amount of fremitus to be obtained over a given part of the thorax varies, of course, according to the loudness of the words spoken, and is influenced also by the vowels contained in them. A certain uniformity is obtained by getting the patient to repeat always the same formula. Thus, he is likely to use the same amount of force each time he repeats them and to use approximately the same pitch of voice.

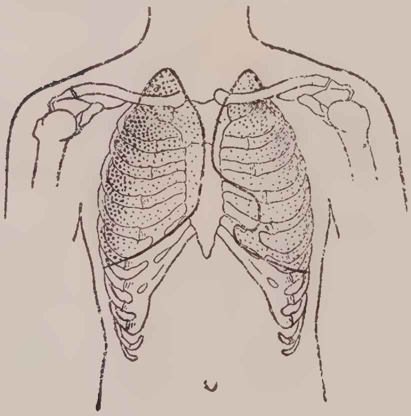

Fig. 81.—Distribution of Tactile Fremitus.

Other things being equal, the fremitus is greater in men than in women, in adults than in children, and is more marked in those whose voices are low pitched than in those whose voices are relatively shrill. The amount of fremitus also varies widely in different parts of the healthy chest. A glance at Fig. 81 will help us to realize this. The parts shaded darkest communicate to the fingers the most marked fremitus, while in the parts not shaded at all, little or no fremitus is felt. Intermediate degrees of vibration are represented by intermediate tints of shading. From this diagram we see at once (*a*) that the maximum of fremitus is to be obtained over the apex of the right lung in front[1], (*b*) that it is greater in the upper part of the chest than in the lower, and somewhat greater throughout the right chest than in corresponding part of the left. *This natural ineuqality of the two sides of the chest cannot be too strongly emphasized.*

Comparatively little fremitus is to be felt over the scapulæ behind, and still less in the precordial region in front. The outlines of the lungs can be quite accurately mapped out by means of the tactile fremitus in adults of low-pitched voice. In children, as has been already mentioned, fremitus is usually very slight and may be entirely absent, and in many women it is too slight to be of any considerable diagnostic value. Again, some very fat persons and those with thick chest walls transmit but little vibration to their chest walls when they speak. On the other hand, in emaciated patients or in those with thin-walled, flexible chests, the amount of fremitus is relatively great.

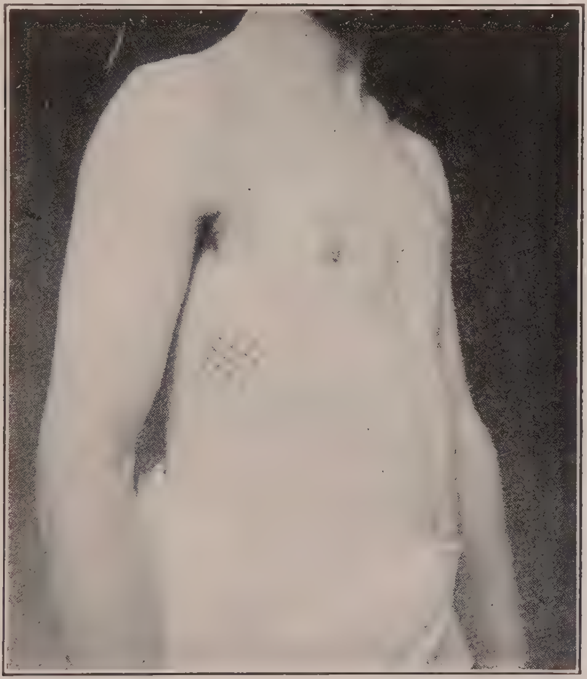

FIG. 82.—Showing Point at Which Pleural Friction is Most Often Heard.

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[1] Owing to the more direct connections through the right bronchus with the larynx.

Bearing in mind all these disparities—disparities both between persons of different age and different sex, and between the two sides of the chest in any one person—we are in a position to appreciate the modifications to which disease gives rise and which may be of great importance in diagnosis. These variations are:

(*a*) Diminution or absence of fremitus.

(*b*) Increase of fremitus.

(*a*) If the lung is pushed away from the chest wall by the presence of *air or fluid or tumor* (pneumothorax, pleurisy, hydrothorax, neoplasms) in the pleural cavity, we get a diminution or absence of tactile fremitus—diminution where the layer of fluid or air is very thin, absence where it is of considerable thickness.

(*b*) Solidification of the lung due to phthisis or pneumonia is the commonest cause of an *increase* in tactile fremitius. Further details as to the variations in amount of fremitus in different diseases may be found in later chapters in this book.

4. *Friction, Pleural or Pericardial*

In many cases of inflammatory roughening of the pleural surfaces ("dry pleurisy"), a grating or rubbing of the two surfaces upon each other may be felt as well as heard during the movements of respiration, and especially at the end of inspiration. Such friction is most often felt at the bottom of the axilla, on one side or the other, where the diaphragmatic pleura is in close apposition with the costal layer (see Fig. 82, p. 99).

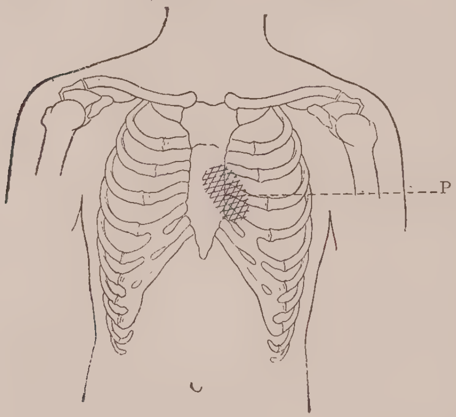

FIG. 83.—Showing Point (P) at Which Pericardial Friction is Most Often Heard.

Similarly, in roughening of the pericardial surfaces ("dry" or "plastic" pericarditis) it is occasionally possible to feel a grating or rubbing in the precordial region more or less synchronous with the hearts movements. Such friction is most often to be felt in the region of the fourth left costal cartilage (see Fig. 83).

Palpable friction is of great value in diagnosis because it is a sign about which we can feel no doubt; as such it frequently confirms our judgment in cases in which the auscultatory signs are less clear. Friction sounds heard with the stethoscope may be closely simulated by the rubbing of the stethoscope upon the skin, but palpable friction is simulated by nothing else, unless occasionally by

5. *Palpable Râles*

Occasionally low pitched, snoring râles communicate a sensation to the hand placed upon the chest in the region beneath which the râles are produced; to the practised hand this sensation is quite different from that produced by pleural friction, although the difference is hard to describe.

6. *Joint Frictions and Tendon Frictions*

F. T. Lord has called attention to grinding, grazing or snapping noises heard over the back and shoulders of patients especially when their arms are crossed on the chest so as to draw the scapulæ forward. Some of these sounds are produced in the shoulder-joint, others in the fascia or muscle-bands about the scapulæ. Since these frictions can be felt as well as heard they are mentioned here. Relaxing all the muscles, and listening over the shoulder-joint, usually prevents our mistaking such sounds for râles or pleural friction.

7. *Local Muscular Spasm*

Pottenger has called attention to the importance of increased tension or rigidity in muscular bundles overlying a focus of tuberculosis in the lung. In some cases this is easily appreciated and may be of value in diagnosis.

8. *Tender Points upon the Thorax*

In mitral disease, dry pleurisy, necrosis of the rib, and some times in phthisis, one finds areas of marked tenderness in different parts of the chest. In mitral disease and "effort syndrome" it is the parts near the apex impulse that are sore.

The tenderness in phthisis is most apt to be in the upper and front portions of the chest. In neurotic individuals we sometimes find a very superficial tenderness over parts of the thorax; in such cases pain is produced by very light pressure, but not by firm pressure at the same point.

The presence of *pulsations* in parts of the chest where normally there should be none is suggested by inspection and confirmed by palpation. It is not necessary to repeat what was said above as to the commonest causes of such abnormal pulsations. When searching for slight, deep-seated pulsation (*e.g.*, from an aortic aneurism), it is well to use bimanual palpation, keeping one hand on the front of the chest and the other over a corresponding area in the back.

The temperature and quality of the skin are often brought to our attention during palpation. After a little practice one can usually judge the temperature within a degree or two simply from the feeling of the skin. Any roughness, dryness, or loss of elasticity of the skin (myxœdema, diabetes, long-standing pyrexia, or wasting disease) is easily appreciated as we pass the hand over the surface of the thorax or down the arms. The same manipulation often brings to our attention in cases of *alcoholism* an unusually *smooth and satiny quality of the cutaneous surface.*

II. The Pulse; Preliminary Study

Fifty years ago the study of the pulse furnished the physician with most of the available evidence regarding the condition of the heart. At present this is not the case. With the increase of our knowledge of the direct physical examination of the heart and of the various methods of measuring the systolic or diastolic pressure on the peripheral arteries, the amount of information furnished exclusively by the pulse proportionately decreased, until within the past ten years when the researches of Wenckebach, the studies of MacKenzie upon the venous pulse, the electrocardiographic work of Einthoven have focussed attention anew upon vascular phenomena as a means of estimating heart function.

Despite the more accurate and detailed information to be obtained by the newer methods, simple manual palpation of the radial pulse is still an important factor in diagnosis, prognosis, and treatment, and will remain so, because it gives us quickly, succinctly, and in almost every case a great deal of valuable information which it would take more time and trouble to obtain in any other way. As we feel the pulse, we get at once a fact of central importance in the case; by the pulse the steps of our subsequent examination are guided. In emergencies or accidents the pulse gives us our bearings and tell us whether or not the patient's condition is one demanding immediate succor—*e.g.*, hypodermic stimulation—and whether the outlook is bright or dark. To gather this same information in any other way would involve losing valuable time.

Again, when one has to see a large number of patients in a short time, as in visiting a hospital ward or on the crowded days of private practice, the pulse is an invaluable short cut to some of the most important data.

Moreover, there are some important inferences which the pulse and *only the pulse* enables us to make. They are not numerous, but their value may be great. Delay in one radical pulse when taken in connection with other signs may furnish decisive evidence of aneurism of the aortic arch; arterial degeneration may betray its presence chiefly in the peripheral arteries.

Since, then, direct palpation of the radial pulse furnishes information of crucial importance in a few diseases, and is a quick, reliable, and convenient indication of the general condition of the circulation in all cases, it is essential that we should study it carefully both in health and in disease.

How to Feel the Pulse

(*a*) We usually feel for the pulse in the radial artery because this is the most superficial vessel which is readily available. Occasionally, as when the wrists are swathed in surgical dressings or tied up in a straight-jacket, we make use of the temporal, facial, or carotid arteries. Hoover believes that the femoral artery is better than the radial when one wishes to judge blood pressure, but this datum seems to me one that should always be secured by instrumental means. In searching for evidence of arterial degeneration the brachial arteries should always be palpated.

(*b*) Both radials should usually be felt at the same time. By making this a routine practice many mistakes are avoided and any difference in the two pulses is appreciated.

(*c*) The tips of three fingers (never the thumb) should be laid upon the artery, and the following points noted:

1. The *rate* of the pulse.
2. The *rhythm* of the pulse.
3. The amount of force necessary to obliterate it (*compressibility*).
4. The *size and shape of the pulse wave.*
5. The *size and position of the artery.*
6. The condition of the *artery walls.*

Each of these points will now be considered in detail.

1. *The Rate of the Pulse*

In the adult male the pulse averages 72 to the minute, in the female 80. In children it is considerably more frequent. At birth it averages about 130, and until the third year it is usually above 100. In some families a slow pulse, 60 or less, is hereditary; on the other hand, it is not very rare to observe a permanent pulse rate of 90 or more in a normal adult (see below, p. 276). Exercise or emotion quickens the pulse very markedly, and after food it is somewhat accelerated. Some account of the causes of pathological quickening or slowing of the pulse will be found on pages 278 and 279.

2. *Rhythm*

The pulse may be irregular in *force*, in *rhythm*, or (as most commonly happens) in both respects. As a rule, irregularities in force are the more serious, whether occurring as *alternation* (see page 123) or in the *absolute* type of arrhythmia which is associated with auricular fibrillation (see below, p. 119). Intermittence or irregularity in rhythm *alone* (respiratory arrhythmia and premature beats) is much less ominous.

Special types of irregularity will be discussed later in connection with the instrumental study of phlebograms and arteriograms.

In general it may be said (*a*) that irregularity in the *force* of the pulse beats is a serious sign, if overexertion and temporary toxic influences (tobacco, tea, etc.) can be ruled out; (*b*) that it is far more serious when occurring in connection with diseases of the aortic valve than in mitral disease; and (*c*) that it often occurs in connection with sclerosis of the coronary arteries and myocardial weakening.

3. *Compressibility, or Systolic Arterial Pressure*

There is no single datum concerning the pulse more important than the amount of force needed to obliterate its beat. Until recently we have had no more accurate method of measuring the systolic blood pressure than that depending on direct digital compression. This method seems to me so unreliable that it should be abandoned in favor of the instrumental method presently to be described.

4. *The Size and Shape of the Pulse Wave*

Of the use of the sphygmograph for representing pulse waves I shall speak later. The points discussed in this section are appreciable to the fingers.

I. The size of the pulse wave—the height to which it lifts the finger—depends on two factors:

(*a*) The force of the cardiac contractions (systolic arterial pressure).

(*b*) The tightness or looseness of the artery (*tension*, or diastolic pressure).

If the arteries are contracted and small, the pulse wave corresponds, while if they are large and relaxed, it needs only a moderate degree of power in the heart to produce a high pulse wave. If the tension remains constant the size of the pulse wave depends on the force of the heart's contraction. If the heart power remains constant, the size of the pulse wave depends on the degree of vascular tension. Vascular tension is estimated in ways to be described presently, and after allowing for it, we are enabled to estimate the power of the heart's contraction from the height of the pulse wave.

II. The *shape of the pulse wave* is also of importance.

(*a*) It may have a very sharp summit, rising and falling back again suddenly; this is known as an *ill-sustained* pulse or a "Corrigan" pulse, and may be due to a lack of sustained propulsive power in the contracting heart muscle, to low vascular tension, diminished vascular elasticity, or to a combination of the three causes. A weak heart with low arterial tension often produces such a pulse wave—deceptively high and giving at first an impression of power in the heart wall, but ill sustained and easily compressible. This is the "bounding pulse" of *early infectious processes*. An exaggeration of this type of pulse is to be felt in *aortic regurgitation* (see page 222) and in many cases of *arteriosclerosis*. Thyrotoxicosis also produces it.

(*b*) In sharp contrast with the above is the pulse wave which lifts the finger gradually and slowly, sustains it for a relatively long period, and then sinks gradually down again. Such a pulse with a "long plateau" instead of a sharp peak is to be felt most distinctly in aortic stenosis, less often in mitral stenosis and other conditions (see page 228).

(*c*) The *dicrotic* pulse wave is one in which the secondary wave, which the sphygmograph shows to be present in the normal pulse, is much exaggerated, so that a distinct "echo" of the primary wave is felt after each beat. If the heart is acting rapidly, this dicrotic wave does not have time to fall before it is interrupted by the primary wave of the next beat, and so appears in the sphygmographic tracing as a part of the up-stroke of the primary wave. This is known as the "*anacrotic* pulse."

(*d*) The shape of the *high-tension pulse wave* will be described in the next section.

5. *Tension, or Diastolic Arterial Pressure*

The degree of contraction of the vascular muscles determines the size of the artery and (to a great extent) the tension of the blood within it. But if the heart is acting feebly, there may be so little blood in the arteries that even when tightly contracted they do not subject the blood within them to any considerable degree of tension.

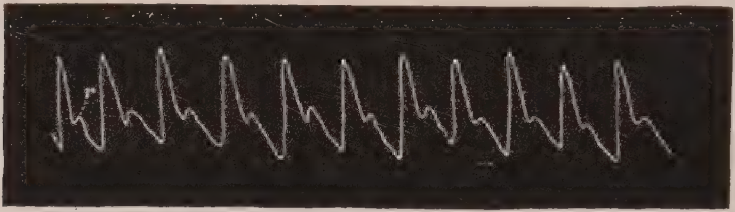

FIG. 84.—Sphygmographic Tracing of Low Tension Pulse.

To produce high tension, then, we need two factors: a certain degree of power in the heart muscle, and contracted arteries. To produce low tension we need only relaxation of the arteries, and the heart may be either strong or weak.

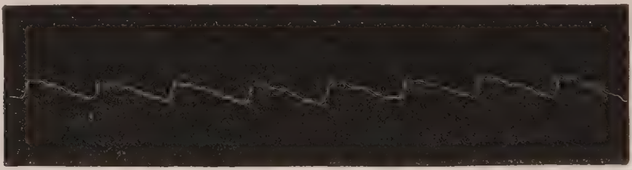

FIG. 85.—Sphygmographic Tracing of High Tension Pulse.

The pulse of low tension collapses between beats, so that the artery is less palpable than usual or cannot be felt at all. Normally, the artery can just be made out between beats, but any considerable lowering of arterial tension makes it altogether impalpable except during the period of the primary wave and of the dicrotic wave, which is often very well marked in pulses of low tension. The shape of the wave under these conditions has already been described (see Fig. 84).

The pulse of high tension is perceptible between beats as a *distinct cord which can be rolled between the fingers*, like one of the tendons of the wrist. It is also difficult to compress in most cases, but this may depend rather on the heart's power than on the degree of vascular tension. A high-tension pulse is often indistinguishable from one stiffened by arteriosclerosis (*vide infra*). The pulse wave is usually of moderate height or low, and falls away slowly with little or no dicrotic wave (see Fig. 85).

6. *The Size and Position of the Artery*

I have often known errors to occur because a small *artery* is mistaken for a small *pulse wave*. The size of the branches of the arterial tree varies a great deal in different individuals of the same weight and height, and if the radial is unusually small and a hurried observation gives us the impression (true, so far as it goes) that there is very little in the way of a pulse to be felt, we are apt to conclude (wrongly, perhaps) that the heart's work is not being properly performed. The effort to obliterate such a pulse, however, *may* set us right by showing that despite the small size of the vessel (and consequently of the pulse wave) it takes as much force as it normally does to obliterate it. But in many cases we can determine the question satisfactorily often by using some instrument for measuring arterial pressure. Thus, a *small pulse wave* (in a congenitally small artery) may be distinguished from a *weak pulse*. From the *contracted* artery of high vascular tension we distinguish the *congenitally small* artery because the latter is not to be rolled beneath the fingers, and is not more than normally palpable between the pulse beats. *Blood pressure measurements, however, are the only reliable guide in such cases.*

Not infrequently the nurse reports in alarm that the patient has no pulse, when in reality the pulse is excellent but the artery misplaced so as to be impalpable in the ordinary situation. It may be simply more deeply set than normal, so that the fingers cannot get at it, or it may run superficially over the end of the radius toward the "anatomical snuff box." Other anomalies are less common. As a rule, the other radial artery is normally placed and can be used as a standard, but occasionally both radials are anomalous and we may be compelled to use the temporal or facial instead.

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[1] "Arterio-sclerosis" is here used to mean any anatomical change in the arterial walls that permanently diminishes their elasticity. No single histological entity is referred to.

7. *The Condition of the Artery Walls*

Arterio-sclerosis is manifested in the peripheral arteries, especially in the brachial, in the following forms:

(*a*) Simple *stiffening* of the arteries without calcification.
(*b*) *Tortuosity* of the arteries.[1]
(*c*) *Calcification.*

Simple stiffening without calcification is due to fibrous thickening of the vessel and produces a condition of the arteries not manually to be distinguished from high tension. The artery can be rolled under the fingers, stands out visibly between the heart's beats, but is not incompressible, has a smooth surface, and is not always tortuous. If it is tortuous as well as stiff, we may conclude that there is an histological degeneration at any rate, whether or not there is increased tension as well. In the majority of cases the two conditions are associated and do not need to be distinguished.

The normal radial artery is straight;[1] hence any deviation is evidence of changes in its walls and is easily recognized as we run our fingers up and down the vessel.

Calcification of an artery produces usually a *beading* of its surface. As we move the fingers along the artery, quickly and with very slight pressure, a series of transverse ridges or beads can be felt. The qualities of the pulse wave within can usually be appreciated fairly well, in this type of artery, but in very advanced cases the calcification is diffuse and converts the radial into a rigid "pipe stem"—absolutely incompressible—unless we break the calcified coat—and easily mistaken for a tendon. In such an artery little or no pulse can be felt.

Such are the points to be noted in a cursory, manual study of the pulse. To enumerate the characteristics of the pulse in the many diseases in which it affords us valuable information is beyond the scope of this book. The qualities to be expected in the pulse in connection with the different diseases of the heart are described in the sections on those diseases. Here it will suffice to enumerate some of the conditions in which vascular tension is usually increased or diminished.

Low tension is produced by moderate exercise, by warmth (*e.g.*, a warm bath), by food. Among pathological conditions we may mention diarrhœa, Addison's disease, pernicious anæmia, many infectious fevers and debilitated states.

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[1] Tortuosity in the temporal artery, however, is normal.

Temporary high tension is produced by *exercise* and *emotion;* also by cold (*e.g.*, cold bathing, malarial chills). Vascular tension is almost always high in toxæmias of pregnancy during and before *eclamptic spasms* and parturition.

Increase of intracranial pressure (as by *cerebral hemorrhage* or *trauma*) has a similar but more lasting effect.

Permanent high tension is due to subacute or chronic nephritis, to arterio-sclerosis or to unknown causes (Allbutt's "Hyperpiesia).

In valvular heart disease without nephritis or arterio-sclerosis the tension is usually normal or slightly lowered.

III. Arterial Pressure and the Instruments for Measuring It

Within the past decade a number of instruments have come into use, the object of which is to tell us with some approach to accuracy the lateral pressure in the peripheral arteries. We have long attempted to estimate this pressure, by simple digital compression and palpation, but today it is recognized universally that digital methods are wholly

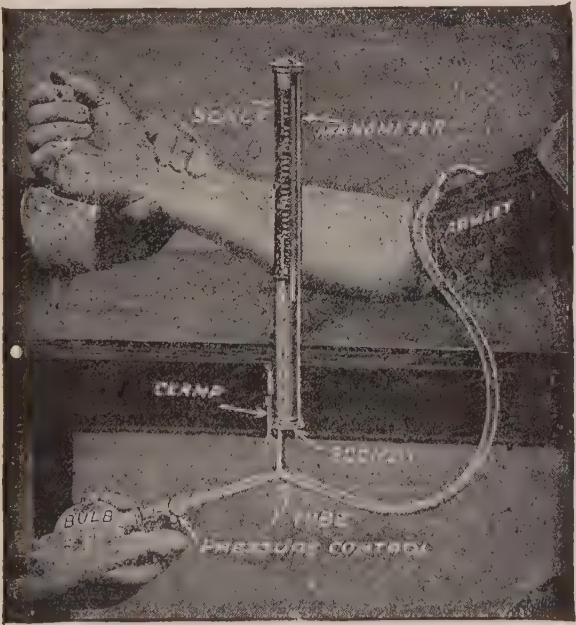

Fig. 86.—Mercer's Type of Riva-Rocci Sphygmomanometer.

unreliable and that only by instrumental methods can we measure arterial tension and so estimate the heart's power.

Here as elsewhere in this book I shall describe only such instruments and methods as seem to me the best. I shall not attempt to cover the whole field or to conceal such personal preferences as are based on experience.

For clinical work instruments of the Riva-Rocci type have now displaced all others in this country) *e.g.*, Nicholson's or the Baumanometer). There are many variations and types all essentially the same and all good so long as a mercury column is their basis. Despite the inconveniences of transporting a mercury column, it remains the most reliable method. Instrument of any other type (*e.g.*, those using springs to measure pressure) are always getting out of order and have to be constantly standardized by comparison with the mercury column.

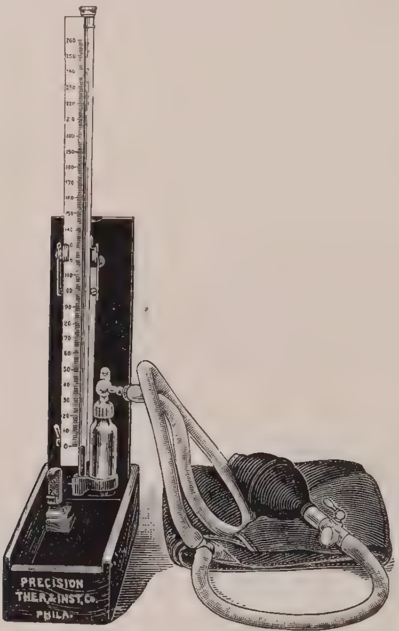

FIG. 87.—Nicholson's Type of Riva-Rocci Sphygmomanometer.

All instruments of the Riva-Rocci type consist essentially of an inflatable rubber armlet, so arranged that it can be fitted closely around the upper arm, a mercury manometer of the ordinary type, and an air-pump (see Figs. 86 and 87). The air forced from the pump is distributed into the rubber armlet and into the manometer at the same time, and experiments have shown that the actual pressure in the armlet is practically identical at any given time with that in the manometer.

1. *Measurement of Systolic Pressure*

(*a*) *Palpatory Method*

Pump in air until the radial pulse stops, and at that instant note the height of the mercury column. The reading thus obtained is taken to represent the systolic or maximum pressure in the brachial artery. To raise the mercury column slightly above the point at which the pulse stops, then let the column slowly fall and note the point on the scale at which we feel the *return of the pulse*, is easier and no less accurate than to fix the point at which the pulse first disappears. This method is inferior to the auscultatory method described below.

It is true that the air within the rubber armlet has to overcome not only the pressure within the radial artery, but the resistance of the artery wall and the elasticity of the soft parts around it. The former factor has been shown to represent a pressure of not more than 2 or 3 mm. Hg, provided the artery walls are normal. If arteriosclerosis is present, it has been estimated by Herringham that the artery may oppose a resistance of 15 to 20 mm. Hg. The more carefully conducted experiments of Janeway, however, convince me that Herringham is wrong and that sclerosed arteries offer a *direct* resistance of less than 5 mm. Hg. The amount of error thus introduced is not of importance.

The resistance of the soft parts around the artery is a factor of no A huge arm gives no higher reading than a shrivelled one, as has been shown by Janeway in a patient one of whose arms was congenitally atrophied while the other was enormous.

(*b*) *Auscultatory Method*

A better way to measure systolic pressure is by listening with the stethoscope over the brachial artery just peripheral to the cuff, and noting when the tapping systolic sound disappears, or better when it first *reappears* as the mercury falls.

2. *Measurement of Diastolic Pressure*

The auscultatory method (Korotkoff) just described is especially useful in the measurement of diastolic or minimum blood-pressure. The air is allowed gradually to leak out of the cuff and, as the mercury column descends, one listens with the stethoscope just below the cuff. The sharp systolic sound reappears, (*record systolic pressure*) becomes loud, and remains so until we reach a point 30–90 mm. below the systolic reading. Then the sound suddenly changes from a sharp tap to a dull, feeble thud. *The height of the mercury column at the instant when this change appears is best taken as the diastolic pressure.* At a point 5 to 10 mm. of mercury below this, the arterial sound disappears altogether and some use this disappearance point to represent the diastolic pressure. The *sudden-weakening point* is, however, more accurate.

3. *Normal Readings*

With any of the various types of Riva-Rocci instrument, the average readings in healthy adults are approximately as follows: Systolic, 110–135 mm. Hg. Diastolic, 60–90 mm. Hg.

The "pulse pressure," *i.e.*, the difference between any individual's systolic and diastolic pressure, averages 30–60 mm. Hg.

In women systolic pressure runs about 10 mm. lower than in men. Children under 2 years 75–90 mm. In older children, 90–110 mm. *Excitement or exercise raises the pressure temporarily but considerably.* It is 5–10 mm. lower in recumbency than in the sitting position.

4. *The Use of the Data Obtained by these Instruments*

Whenever it is important for us to know the tension of the peripheral arteries, a sphygmomanometer is indispensable. The Riva-Rocci instrument as modified by Nicholson and others has now secured a firm position in the routine work of good clinicians all over this country.

(*a*) *High Systolic Blood-pressure*

Slight increase of systolic blood-pressure is (fortunately for our diagnosis) rarely encountered. As a rule if we find blood pressure high it is very high, obviously high, 160–250 mm. Hg., and not in the more dubious immediate regions (140–160). In at least 95 per cent. of the cases a permanent or long standing hypertension is associated with obvious hypertrophy of the heart and due to *all the well known causes of cardiac hypertrophy except rheumatic endocarditis with valve lesions.* Accordingly the types of high systolic blood pressure are those due to:

1. *Unknown cause* (*Allbutt's "Hyperpiesia"*).
2. *Chronic nephritis.*[1]
3. *Arterio-sclerosis.*[2]
4. *Syphilitic aortitis with aortic regurgitation.*
5. *Thyrotoxicosis* (Graves' disease).

Less important are the transitory effects of puerperal autointoxication, of acute brain-pressure as in apoplexy, skull fractures, and meningitis; also of acute pain (as in gout, tabetic crises, lead colic, biliary colic and during child birth).

Clinically it is easy to exclude in most cases all the causes of hypertension just listed *except three: "primary" or toxic hypertension* (Allbutt), *chronic nephritis*, and *arterio-sclerosis. Hence vascular hypertension and high blood-pressure should always make us suspect these diseases even though the urine and the peripheral arteries give no convincing sign of them.*

We may thus detect in life insurance examinations and elsewhere many cases that would otherwise pass altogether unnoticed. On the other hand the negative value of a normal blood-pressure is very great. It helps us to exclude chronic nephritis in the vast majority of cases, and except in the aged raises a presumption against the existence of arterio-sclerosis.

(*b*) *Low Systolic Blood-pressure*

Much less valuable in diagnosis are the abnormally low blood-pressure readings. We seldom get much help from them. The lowest readings occur in Addison's disease (even to 50 mm.), after hæmorrhage, infectious fevers (tuberculosis, pneumonia, typhoid) and surgical "shock." Occasionally attacks of faintness, vertigo, headache and muscular debility are associated (especially in young adults) with a hypotension of unknown origin.

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[1] *i.e.*, any nephritis of more than a few weeks duration.

[2] Especially if the thoracic, aortic and splanchnic arteries are extensively diseased.

(*c*) *Diastolic Pressure*

The conclusions about the strength of the heart muscle recently drawn by several writers from formulæ based on pulse pressure and its relations to systolic and diastolic pressure seem to me to rest on quite uncertain theoretic foundations. They are not verified by clinical observation or by autopsy results.

Diastolic pressure should be measured in every case showing high systolic pressure, and when we have learned the technique it is about as easy to measure both systolic and diastolic pressure by the auscultatory method and in every case. The high systolic pressures of aortic regurgitation is rendered relatively unimportant by the correspondingly depressed diastolic pressure. The same is true in many cases of arterio-sclerosis and hyperthyroidism.

High systolic pressures as measured in office patients or in any anxious or excited person are often temporary and significant of nothing except the patient's state of mind. *But the particular value of measuring diastolic pressure* is that it is influenced little or not at all by emotional states and remains low even when the systolic pressure is raised by emotion to 140 or even higher.

Moreover in some cases of pathological and serious hypertension it is chiefly the diastolic pressure that is elevated, *e.g.*, systolic only 160 —diastolic 120.

On the whole, then, diastolic pressure is decidedly more important than systolic pressure in diagnosis. A permanently high diastolic pressure (over 100 mm. Hg.) is almost always due to nephritis, arteriosclerosis or hyperpiesia, while a permanently or temporarily high systolic pressure (140–160) may mean very little.

Systolic pressures above 160 are usually associated by high diastolic pressures. Aortic regurgitation is the only exception of importance, though in thyrotoxicosis and arteriosclerosis we may find the same thing.

CHAPTER VI

CARDIAC PHYSIOLOGY AND PATHOLOGY AS REVEALED BY ARTERIOGRAMS, PHLEBOGRAMS, AND ELECTROCARDIOGRAMS

In the early editions of this book I referred to the sphygmograph as a fascinating but useless little toy. As an instrument for recording the *shape* of pulse waves it went out of use, because it was hopelessly unreliable. To record the *time relations* of the pulse waves it has come back into use, so that Lewis, one of the most distinguished investigators in the field of phlebographic and electrocardiographic work, is able to state, in his recent monograph[1] that "*the mechanism of the heart may be identified in the majority of cases in which it is irregular by a careful examination of the radial pulse tracing alone.*"

We may come back to the sphygmograph and to the results obtained through it, and, interpreting our old data in the light of newer researches, we may be able to read far deeper and more intelligible meanings into them. But whether this is true or not I feel convinced by my own experience with tracings of the venous pulse and by my study of other's work with electrocardiograms that neither method is likely ever to be used by the practitioner for whom this book is intended. I desire therefore, in this chapter, to state the *results* accomplished by the newer methods without attempting to describe or recommend the technique.

The advances achieved through phlebographic and electrocardiographic work may be summed in a *classification of the arrhythmias* or better as a new grouping of the disturbances in the heart beat.

1. Heart block, partial or total.
2. Auricular fibrillation and absolute irregularity of the pulse.
3. Paroxysmal tachycardia and auricular flutter.
4. Premature contraction of auricle or ventricle.
5. Alternation.

Paul D. White's article in the *Amer. Journ. Med. Sc.*, June, 1915, gives us an interesting statistical picture of these disturbances and serves as a table of contents, a classification and rough estimate of the size of each class. At the end of eight months work at the Mass. General Hospital, White's results could be compressed into the following table.

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[1] Thomas Lewis: "The Mechanism of the Heart Beat," Shaw & Sons, London, 1911, a work to which I am profoundly indebted.

Disturbances of the Heart Beat Found in 300 Cases of Cardio-vascular Disease (Paul D. White)

1. (*a*) Ventricular premature beats in	119 cases.
(*b*) Auricular premature beats in	22 cases.
2. Auricular fibrillation in	71 cases.
3. Alternation in	71 cases.
4. Delayed conduction and heart block in	22 cases.
5. Paroxysmal tachycardia	9 cases.
Auricular flutter	6 cases.
6. Sinus arrhythmia (Disturbances of impulse production). No records kept.	

Blashford & Willis (Arch. of Int. Med., Jan., 1918), found: In 3500 electrocardiograms

1. Auricular fibrillation	363 cases.
2. Ventricular premature beats	316 cases.
3. Auricular premature beats	160 cases.
4. Paroxysmal tachycardia	5 cases.
5. Auricular flutter	9 cases.

The meaning of these terms will next be explained, though the order of numerical frequency will no longer be followed.

I. Heart Block

In the normal heart the impulse of contraction starts at the *sino-auricular node*, a mass of specialized muscular tissue at the superior cavo-auricular junction, spreads through the *bundle* of *His* in the membranous interventricular septum, and is distributed thence through the Purkinje fibres to the ventricles and their papillary muscles. Since the sino-auricular node just mentioned is apparently responsible for the organization of beat after beat, and determines the rate of the heart, it is now often spoken of as "the pace-maker."

If the transmission of impulses is blocked by disease in the bundle of His (usually gumma or fibro-calcareous degeneration), we get the following series of disasters, each worse than the last:

(*a*) A prolongation of the interval between auricular systole and ventricular systole ("the A-s—V-s interval," normaly from .1 to .2 of a second) to twice or thrice its normal length.

(*b*) "*Dropped beats*," *i.e.*, an occasional "*silence*" of the ventricle in answer to the regular auricular contraction preceding it.

(*c*) Regularly recurring "dropped beats" every tenth beat or oftener.

(*d*) The establishment of a 3:1 or 2:1 rhythm *i.e.*, three or two beats of the auricle for every beat of the ventricle.

(*e*) Complete dissociation of auricle and ventricle. No impulses pass down. The ventricle may stand still for good and all or may gradually initiate a slow rhythm of its own (approximately 32 per minute).

If the ventricular silence lasts three to five seconds the patient usually loses consciousness for a moment. Silence of ten to twenty seconds usually results in epileptiform convulsions. When these cerebral phenomena are associated with more or less heart block we have the *Stokes-Adams syndrome*. Silence over ninety seconds means death. (Lewis, *loc. cit.*, p. 266.)

Heart block in slight grades (see (*a*) above) is often a result of "rheumatic" infection of the heart, especially (as Mackenzie and Lewis have shown) in cases of *mitral stenosis*. Lewis found an A-s—V-s interval of over .2 seconds in nearly 17 per cent. of 84 cases of comparatively well-compensated out-patient cases of mitral stenosis. In ward patients it is one of the rarest types of arrhythmia. In White's series it was found but 22 times among 300 cases of cardiovascular disease.

When the A-s—V-s interval is already increased, the act of swallowing, pressure on the vagus in the neck or the administration of digitalis may increase the grade of heartblock, and may lead to a 2:1 to 3:1 rhythm, or to complete dissociation of auricle and ventricle, always a very serious condition.

To diagnose heart block we need electrocardiograms or venous pulse tracings made synchronously with arterial pulse tracings, so that the time relation of auricular contraction (shown in the neck veins) to ventricular contraction (shown in the carotid or radial artery) can be demonstrated. Mackenzie's polygraph records upon the same strip of paper the movements of the auricle (neck vein), of the ventricle (radial pulse or carotid), and of a time marker from which we can calculate very exactly any delay in the passage of the contraction wave from auricle to ventricle. Sometimes we can see in the neck or hear with the stethoscope[1] something corresponding to the auricle movements, and, comparing these facts with the palpable radial impulse, may recognize the severer grades of heart block, but no certainty can be obtained without the use of venous pulse tracings or electrocardiograms. In the latter we have a record of the electrical changes corresponding to the earliest contraction wave in the auricles and in the ventricles respectively—the two waves recorded in the same tracing so that no time comparisons or measurements of different curves are necessary.

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[1] Stokes noted that the dropped beats (auricular movements) in heart block produced "small sounds which may give the illusion of reduplication of either sound." Hirschfelder describes them as "very soft distant sounds like the ticking of a watch" and "best heard along the left sternal margin."

Luckily for those who cannot get the time and money necessary for the use of these methods, there are relatively few cases in which it is imperatively necessary for any purpose of diagnosis, prognosis, or treatment that we should recognize heart block. Were it a commoner or a more curable condition the general practitioner would sometimes be at a great disadvantage. As it is, we can often recognize and treat *the disease underlying heart block* with about the same measure of success whether the heart block itself escapes us or not; but in *heart block itself* there is sometimes effective treatment by thyroid extract, which tends to decrease the grade of block and increases the idio-ventricular rate.

In 3219 cases electrocardiographed at the Massachusetts General Hospital between October 21, 1914 and March 15, 1922, the following figures were obtained by Paul D. White:

	Cases		Per Cent.	
Auriculoventricular block,				
Complete	27		0.8	
Partial	129	156	4.0	4.8
Intraventricular block,				
Bundle branch	41		1.2	
Lesser degree ("aberration")	89	130	2.8	4.0
All cases showing auriculoventricular or intraventricular block		252		7.8
"Sinoauricular block"		11		0.3

Dr. White adds the following definitions:

Complete auriculoventricular block consists of complete dissociation between the auricular and ventricular contractions, with an idioventricular rate of sixty or less.

Partial auriculoventricular block consists of partial dissociation between the auricular and ventricular activity, in which the P-R interval is abnormally long (.2 second or over in this series), or in which there are dropped beats, and where ventricular escape is not primarily the cause of the dissociation.

Intraventricular block is the expression used for delay or blocking in the arborizations of the auriculoventricular conduction system below the bifurcation of the bundle of His.

"*Sinoauricular block*" is that condition of the cardiac mechanism in which the sinoauricular node fails to function either at intervals or continuously.

In White's opinion intraventricular block is of greater significance than auriculoventricular block. To detect the former one needs the electrocardiograph.[1]

II. Auricular Fibrillation

The researches of Mackenzie in 1904–5 brought out the fact that when a heart is *absolutely and perpetually irregular*, *i.e.*, when no two successive beats have the same duration (as shown by a radial pulse tracing), the venous pulse tracing shows no sign of the normal auricular wave, but corresponds with the ventricular systole. This was thought at first to be due to auricular paralysis, but further electrocardiographic research has brought convincing evidence that the auricle is in fact fibrillating, *i.e.*, the seat of innumerable, incoördinate contractions, each limited to a small bundle of muscle fibres. The fibrillating auricle is quite ineffectual and remains in the diastolic position. This condition has long been familiar in animal experimentation, but has not until the last few years been capable of recognition in man.

Though venous pulse tracings and electrocardiographic records are necessary for the absolute demonstration of auricular fibrillation in any case, the presence of an absolutely irregular pulse (*delirium cordis*) is practically equivalent to proof of auricular fibrillation. The exceptions to this rule are negligible. Since the auricles are of use chiefly as temporary reservoirs, the circulation can and does go on for months and years despite the absence of any effectual contraction of the auricle. The ventricle and the peripheral arteries do the work, while the quivering, distended auricle showers impulses at the upper end of His' bundle. "From this turmoil of the auricle a rapid and haphazard succession of waves escapes"[2] along the bundle and produces the absolutely irregular pulse.

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[1] Paul D. White: Observations on Heart Block, in Transactions of the Association of American Physicians, 1922.

[2] Lewis, *loc. cit.*, p. 247.

Auricular fibrillation thus defined produces the great majority of all the arrhythmias clinically observed in the failing heart. The irregular pulse of most cases of rheumatic endocarditis and of myocardial insufficiency (the familiar arteriosclerotic or cardiorenal types) is of this kind.

Auricular fibrillation usually lasts as long as the period of failing compensation which it accompanies. But the auricles may go on fibrillating even after compensation is restored.

Occasionally short flights of fibrillation occur with normal rhythm before and after them ("paroxysmal fibrillation").

III. Paroxysmal Tachycardia

Excluding the periods of accelerated heart action due to emotional strain, muscular exertion and infectious disease, we have but one condition in which the heart suddenly becomes rapid, without irregularity and without previous evidence of cardiac disease. This condition, long known as *paroxysmal tachycardia*, has been illuminated by recent researches. It is a relatively rare condition, occurring nine times among 300 cases investigated by White (see table, p. 116). We now know, through electrocardiographic investigations, that while in the vast majority of these tachycardias the cardiac impulse comes down from the auricle in the ordinary way, it does not arise at the ordinary place, *i.e.*, at the pace-maker, but starts up like an insurrection at some other point in the auricular muscle ("heterogeneous impulses"). Rarely does such a tachycardia originate in the ventricle or in the bundle of His. The present condition of our knowledge is fairly represented by the *diagram from Lewis* (*loc. cit.*, p. 191) reproduced on page 122.

This diagram suggests the "close relation" of paroxysmal tachycardia to the more serious auricular fibrillation just described and to the commoner and less serious forms of arrhythmia due to premature beats.

Histologically nothing definite is known about the cause of paroxysmal tachycardia.

1. *Auricular Flutter*

When the auricular rate rises above 200 but remains regular, while the ventricle beats at a slower rate (usually ½) owing to heart block, the term flutter is arbitrarily applied. It occurs in cases of toxic goitre and of mitral stenosis or other "rheumatic" forms of heart disease. It may occur despite good compensation and is not in itself of much importance. Its import depends on what else is wrong with the heart. Chronic cases may be cured by large doses of digitalis.

IV. Premature Beats (Extrasystoles)

1. *Ventricular.*—Of all disturbances of rhythm (excluding the semi-physiological sinus arrhythmia) ventricular premature beats are the commonest. Paul White[1] found them in 119 out of 300 cases of cardiac disease examined within eight months at the Mass. General Hospital. In the decompensated cases it is relatively rare (10 per cent.) compared to fibrillation and alternation. Like many of the newly discovered cardiac anomalies, ventricular escape is comparable to insubordination or civil war in the heart. It is recognized:

(*a*) In part by the fact that the pause following it in the arteriogram is "compensatory," *i.e.*, just makes up for the shortening of the pause before the premature beat.

(*b*) Because such premature beats are usually weak and barely palpable at the wrist, while the succeeding beat is unusually strong—another form of compensation.

(*c*) By auscultation; the second sound is often found to be absent, only the first sound is heard.

The ventricular origin of the contraction is shown by the fact that the venous curve from the jugular exhibits a regular uninterrupted rhythm of auricular waves in the tracing.

2. *Auricular.*—Premature contractions arising in the auricle and leading to a similarly premature ventricular beat are less commonly seen than "ventricular extrasystoles." In White's series there were but 22 cases as compared with 119 of the ventricular type. The pause following such a beat may be compensatory in the sense explained above, but *usually it is not so.*

The electrocardiogram shows that these abnormal beats do not arise at the pace-maker, but from some other part of the auricle—another example of cardiac civil war or uncoördinated independent action by a portion of the heart muscle. In the same way the ventricular extrasystoles are shown to arise from some abnormally active bit of ventricular muscle.

3. The clinical significance of premature beats is not yet clear. Mackenzie believes that they have little or no importance either in

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[1] Paul D. White: Alternation: *Am. Journ. of Med. Sciences*, June, 1915.

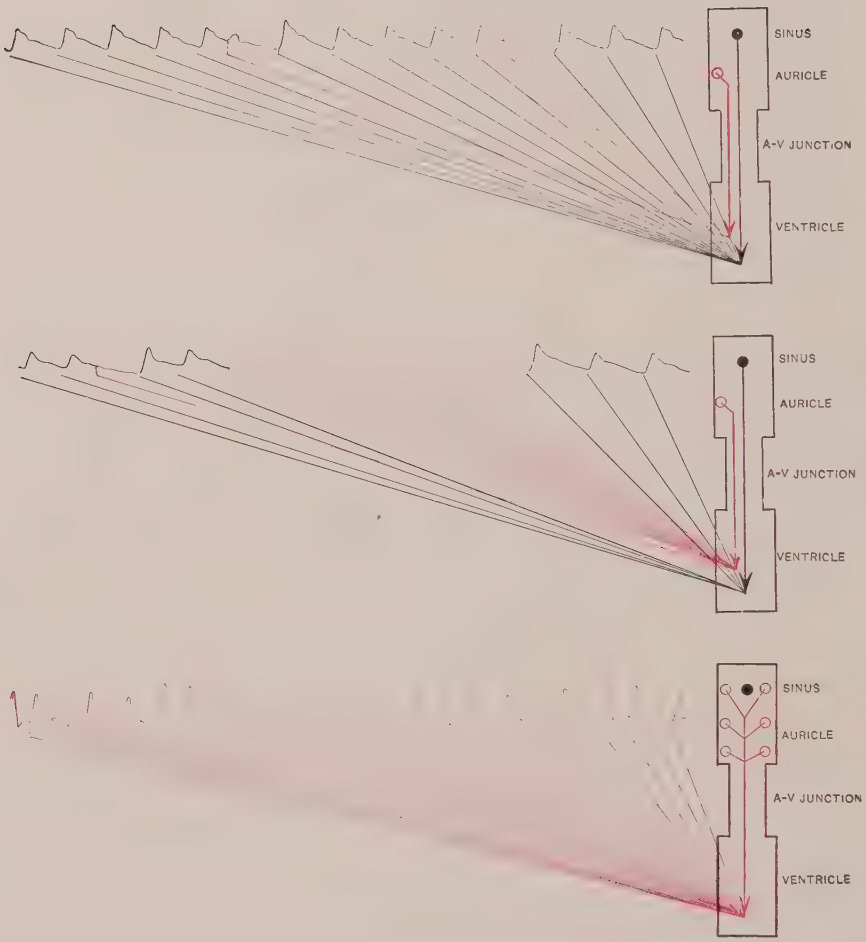

Fig. 88.—(After Lewis.)

I. Occasional premature beats ("auricular extrasystoles"): Indicated in red.
II. A short period of paroxysmal tachycardia. Indicated in red.
III. Absolute arrhythmia (auricular fibrillation).

Lewis' experiments indicate that these three disturbances represent three increasing degrees of typical impulse-formation, "all stages of one or the same process" of disorder or civil war in the heart muscle.

diagnosis or prognosis. In many cases they certainly represent one of the mildest and most harmless types of arrhythmia, but whether this is always true is not yet clear. Their significance probably depends on the associated anomalies discoverable in the circulation. If the heart is sound in every other respect, premature beats have no considerable importance. If, however, hypertension or valvular lesions are also present the "extrasystole" may be an early sign of failing compensation.

V. Coupling of the Heart Beats

In uncompensated heart disease of any type, when the auricle is fibrillating, coupling of the ventricular beats often follows the administration of digitalis. Indeed, in the vast majority of cases we can assume that the patient has recently been taking digitalis if we find the beats coming in close-knit pairs with pauses of varying lengths between the pairs. Other types of coupling also occur, but will not be mentioned here.

1. *Alternation*

In the healthy but overtaxed and rapidly beating heart, in many cases of paroxysmal tachycardia, and in the myocardial insufficiency of the senile heart, one may find a regular alternation of strong and weak beats with or without a disturbance of rhythm. An auricular beat precedes each ventricular beat in normal fashion.

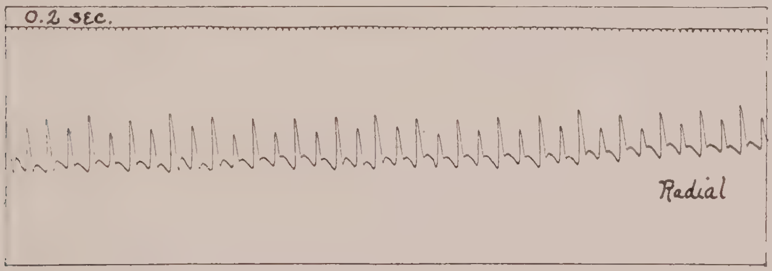

Fig. 89.—Radial Tracing in a Case of Alternation. (Paul D. White.)

The weak beat appears in the arteriogram (see Fig. 89). It may also be noticed while taking the blood-pressure by the auscultatory method: as one slowly lets the mercury column fall one may suddenly begin to hear a doubled rate in the tapping sounds, as the smaller waves begin to come through. Thereafter for a few beats one may hear that every other beat is accented.

When permanent, alternation is always a serious symptom and means that "the heart muscle is in a precarious condition, be it structural or functional." (Lewis, *loc. cit.*, p. 278.)

Immediately following a premature beat, a few alternating beats may often be recognized, as has recently been shown by Paul White. In these cases alternation is not always of serious import. White's studies revealed 71 cases of alternation among 300 patients with cardiovascular disease. But in 79 per cent. the alternation was inconstant and occurred only for a few beats following premature contractions. *Only in 15 out of 71 cases (21 per cent.) was the alternation constant*, and so ominous.

VI. Sinus Arrhythmia

I include here, for convenience, a cardiac irregularity which is not pathological. It is supposedly due to change in vagal tone and is present in some degree in most sound young adults and many older people. The cardiac contractions take place in the normal way, but their rate waxes and wanes—usually quickening with inspiration and slowing with expiration, occasionally without any such connection.

VII. Ventricular Preponderance

Besides the arrhythmias and defects of contraction, an excessive contractile force either of the left ventricle or of the right appears in some of the electrocardiograms. This tells us which ventricle is the larger and so helps in some doubtful diagnoses. For instance, between mitral stenosis and goitre heart, right sided predominance in the electrocardiogram favors stenosis.

CHAPTER VII

PERCUSSION

I. TECHNIQUE

There is no other method of physical examination which needs so much practice as percussion, and none that is so seldom thoroughly learned. Many physicians never succeed in acquiring a facility in the use of it sufficient to make them rely upon their results. Undoubtedly one of the greatest difficulties arises from the necessity of being at once active and passive—at once the percussor and the one who listens to the percussion. Students half unconsciously get to treat the percussion as an end in itself, and hammer away industriously without realizing that two-thirds of the attention must be given to listening, while the percussion itself should become semi-automatic.

It is undoubtedly an advantage to possess a musical ear, but this is by no means a necessity. Some of the most accurate percussors that I know possess absolutely no musical ear—no ear, that is, for pitch—and form their judgments in percussing upon the quality or intensity of the note, and upon the sense of resistance.

In this country practically all percussion is done with the fingers; in Europe instruments are still used to a considerable extent.

I. *Mediate and Immediate Percussion*

Percussion may be either "mediate" or "immediate," the latter term referring to blows struck directly upon the chest with the flat of the hand, or upon the clavicles with the tip of the second finger.

(*a*) *Methods*

Mediate percussion (which is used ninety-nine hundredths of the time) is performed as follows:

The patient should either lie down or sit with his back against some support. The reason of this is that for good percussion one needs to press very firmly with the middle finger of the left hand upon the surface of the chest, so firmly that if the patient is sitting upon a stool without support for his back, it will need considerable exertion upon his part to avoid losing his balance.

In percussing the front of the chest it is important to have the patient sitting or lying *in a symmetrical position*—that is, without any twist or tilting to one side. His head should point straight forward and *his muscles must be thoroughly relaxed.* Many patients, when stripped for examination, swell out their chests and sit up with a military erectness. The muscular tension thus produced modifies the percussion note and causes an embarrassing multitude of muscle sounds which greatly disturb auscultation. Some muscular tensions which produce dulness are probably due to reflex stimulation from disease within the chest and cannot be voluntarily relaxed.

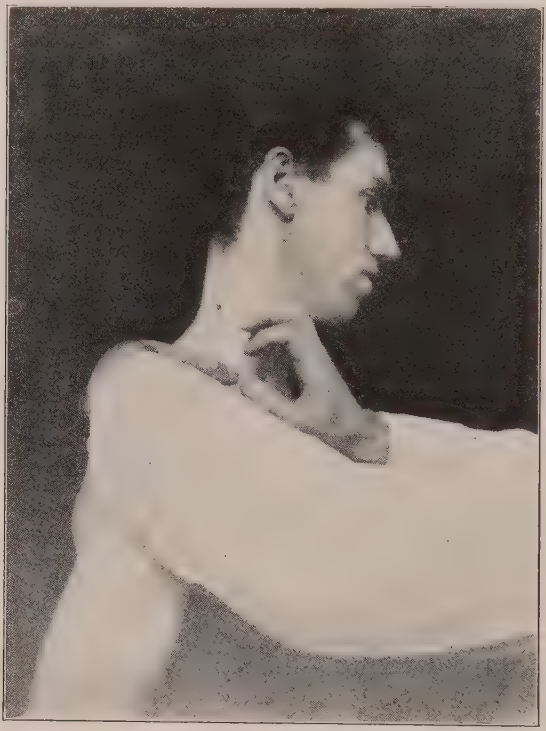

FIG. 90.—Position of the Hands When Percussing the Right Apex.

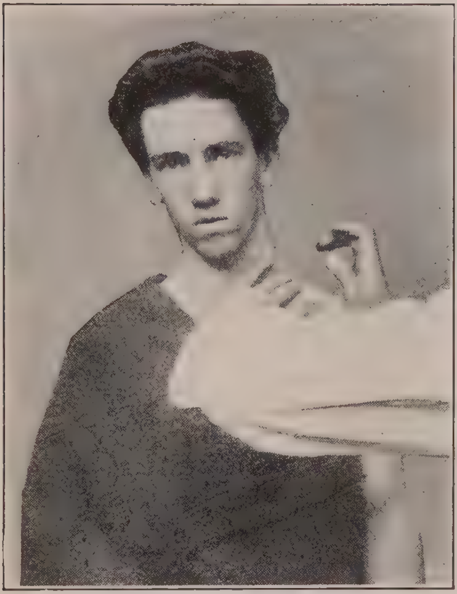

FIG. 91.—Position of the Hands When Percussing the Left Apex.

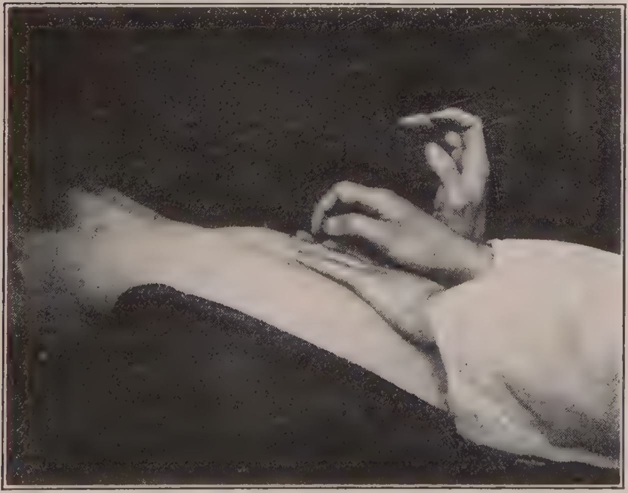

FIG. 92.—The Right Way to Percuss—*i.e.*, From the Wrist.

Having placed the patient in an easy and symmetrical position, our percussion should proceed according to the following rules:

(1) Always press as firmly as possible upon the surface of the chest with the second finger of the left hand[1] on the dorsum of which the blow is to be struck. Raise the other fingers of the left hand from the chest so as not to interfere with its vibrations.

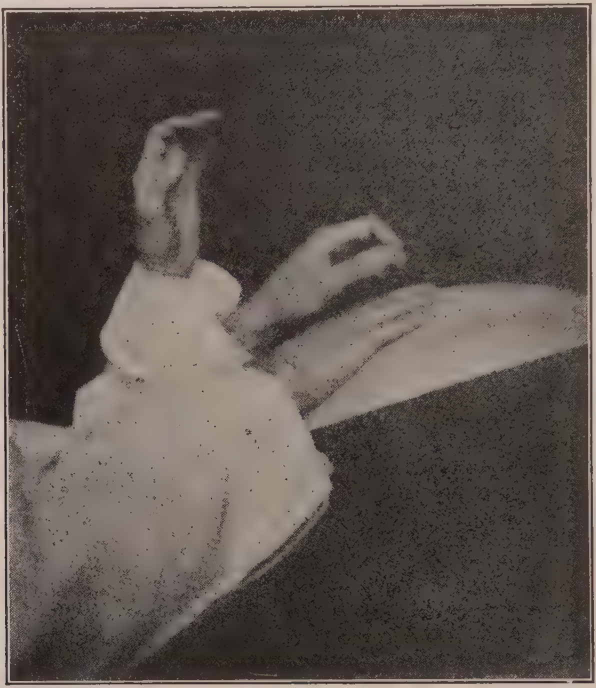

FIG. 93.—The Wrong Way to Percuss—*i.e.*, From the Elbow.

(2) Strike a quick, perpendicular, rebounding blow with the tip of the second finger[2] of the right hand upon the second finger of the left just behind the nail, imitating as far as possible with the right hand the action of a piano-hammer. The quicker the percussing finger gets away again after striking, the clearer will be the note obtained.

[1] Left-handed percussors will, of course, keep the right hand upon the chest and strike with the left.

[2] When percussing the right apex I prefer to strike upon the thumb (see Figs. 96 and 97) as it is almost impossible when standing directly in front of the patient to fit any of the fingers comfortably into the right supraclavicular fossa.

(3) Let all the blows struck in any one part of the chest be uniform in force.

(4) Strike from the wrist and not from the elbow (see Figs. 92 and 93). The wrist must be held perfectly loose.

(5) Keep the percussing finger bent at a right angle as in Fig. 94.

The force to be used in percussion depends upon the purpose for which the percussion is used—that is, upon what organ we are percussing—and also upon the thickness of the muscles covering that part of the chest. For example, it is necessary to percuss very strongly when examining the back of a muscular man, where an inch or two of muscle intervenes between the finger on which we strike and the lung from which we desire to elicit a sound. Over the front of the chest and in the axillæ the muscular covering is much thinner, and hence a lighter blow sufficies. In children or emaciated patients, or in any case in which the muscular development is slight, percussion should be as light as is sufficient to elicit a clear sound. Heavy percussion is sometimes necessary but always unsatisfactory, in that the sound which it elicits comes from a relatively large area

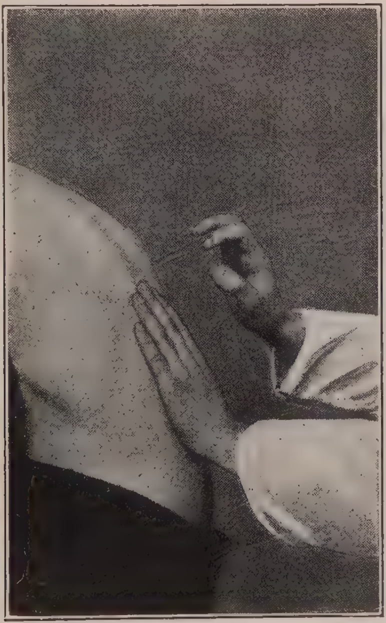

Fig. 94.—Proper Position of the Right Hand During Percussion.

of the chest and does not therefore give us information about the condition of any sharply localized area. If a carpenter, in tapping the wall to find the position of the studs, strikes too hard, he will fail to find the beam, because the blow delivered over the spot behind which the beam is situated is so forcible as to bring out the resonance of the hollow parts around. It is the same with medical percussion. Heavy percussion is always inaccurate. It may be necessary where the muscles are very thick, but its value is then proportionately diminished. On the other hand, it is possible to strike so lightly that no recognizable sound is elicited at all. The best percussion, therefore, is that which is just forcible enough to elicit a clear sound without setting a large area of chest wall in vibration.

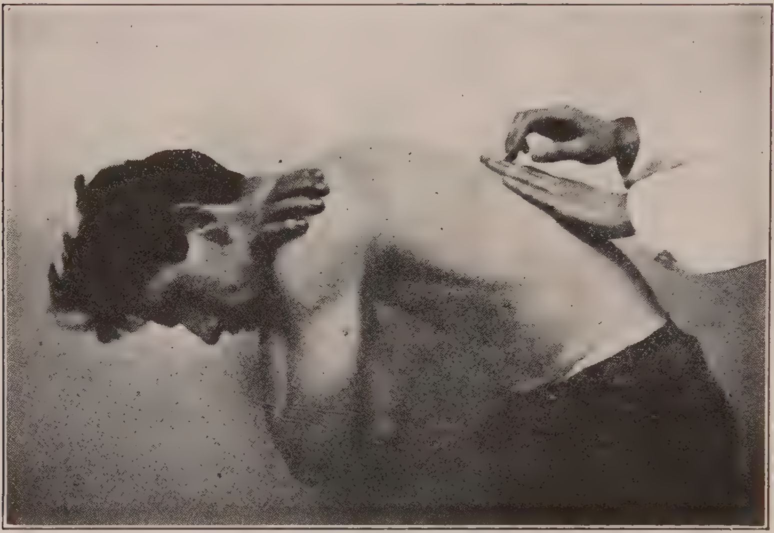

FIG. 95.—Proper Position of the Patient During Percussion of the Back.

When we desire to percuss the back, it is important to get the scapulæ out of the way as far as possible. To accomplish this, we put the patient in the position shown in Fig. 95, the arms crossed upon the chest. This position sometimes produces crackling or grazing sounds transmitted from the shoulder-joint or from the taut muscle-bands. In case of doubt the hands should be dropped loosely at the sides (F. T. Lord). The patient should be made to bend forward; otherwise the left hand of the percussor will be uncomfortably bent backward and his attention thereby distracted (see Fig. 96).

When the axillæ are to be percussed, the patient should put the hands upon the top of the head.

2. *Auscultatory Percussion*

If while percussing one auscults at the same time, letting the chest piece of the stethoscope rest upon the chest, or getting the patient or an assistant to hold it there, the sounds produced by percussion are greatly intensified, and changes in their volume, pitch, or quality are very readily appreciated. The blows must be very lightly struck, either upon the chest itself or upon the finger used as a pleximeter in the ordinary way. Some observers use a short stroking or scratching touch upon the chest itself without employing any pleximeter.

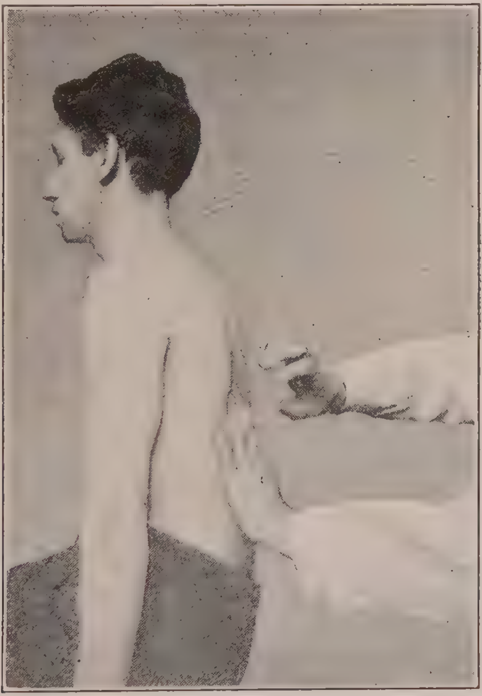

FIG. 96.—Wrong Position for Percussing the Back. The patient should be bent forward.

This method is used especially in attempting to map out the borders of the heart and in marking the outlines of the stomach. In the hands of skilled observers it often yields valuable results, but one source of error must be especially guarded against. *The line along which we percuss, when approaching an organ whose borders we desire to mark out, must neither approach the chest piece of the stethoscope nor recede from it.* In other words, the line along which we percuss must always describe a segment of a circle whose centre is the chest piece of the stethoscope (see Fig. 97). If we percuss, as we ordinarily do, in straight lines toward or away from the border of an organ, our results are wholly unreliable since every straight line must bring the point percussed either closer to the stethoscope or farther from it, and the intensity and quality of the sounds conducted through the instrument to our ears vary directly with its distance from the point percussed.

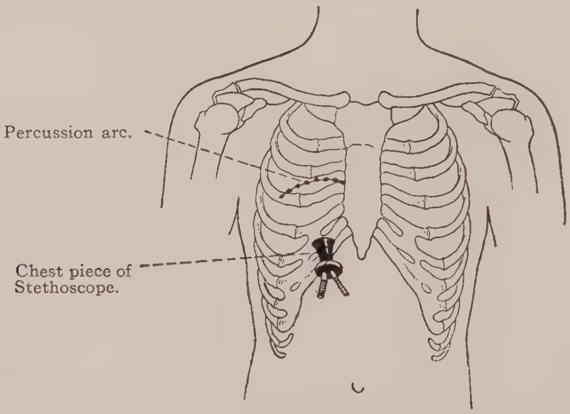

Fig. 97.—Auscultatory Percussion, Showing the Arc along which such Percussion should be made.

It will be readily seen that the usefulness of auscultatory percussion is limited by this source of error, and that considerable practice is necessary before one can get the best results from this method. Nevertheless it has, I believe, a place, though not an important one, among serviceable methods of physical examination.

3. *Palpatory Percussion*

Some German observers use a method of percussion in which attention is fixed directly or primarily on the amount of resistance offered by the tissues over which percussion is made. Even in ordinary percussion the amount of resistance is always noted by experienced percussors, but the element in sound is usually the main object of attention. Palpatory percussion is rather a series of short *pushes* against various points on the chest wall, but some sound is elicited and probably enters into the rather complex judgment which follows.

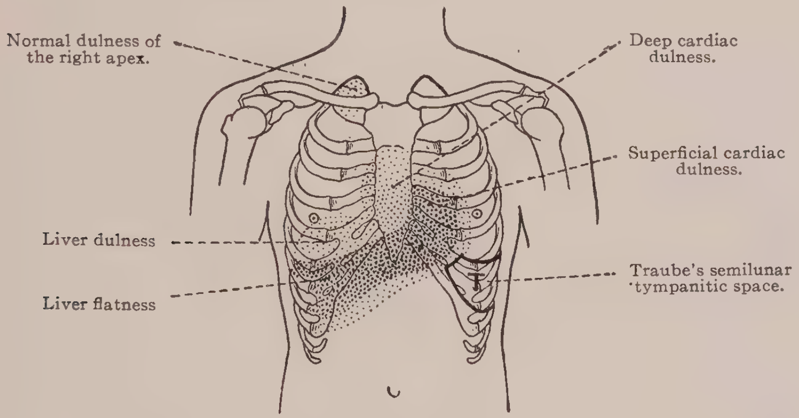
Normal dulness of the right apex.
Deep cardiac dulness.
Superficial cardiac dulness.
Liver dulness
Liver flatness
Traube's semilunar tympanitic space.

FIG. 98.—Percussion Outlines in the Normal Chest.

In this country palpatory percussion is but little employed.

Some physicians use a bit of flexible rubber for direct percussion, but I have seen no advantage gained by it.

II. Percussion Resonance of the Normal Chest

The note obtained by percussing the normal chest varies a great deal in different areas. In Fig. 98, the parts shaded darkest are those that normally give least sound when percussed in the manner described above, while from the lightest areas the loudest and clearest sound may be elicited.

1. *Vesicular Resonance*

The sound elicited in the latter areas is known as normal or "*vesicular*" resonance, and is due to the presence of a normal amount of air in the vesicles of the lung underneath. If this air-containing lung is replaced by a fluid or solid medium, as in pleuritic effusion or pneumonia, it is much more difficult to elicit a sound, and such sound as is produced is short, high pitched, and has a feeble carrying power when compared with the sound elicited from the normal lung.

This short, feeble, high-pitched sound is known technically as a "*dull*" or "*flat*" sound, flatness designating the extreme of the qualities that characterize dulness. Over the parts shaded dark in Fig. 98, we normally get a dull or flat tone, the darkest portions being flat and the others dull. The heavy shadow on the right corresponds to the position occupied by the liver, or rather by that part of it which is in immediate contact with the chest wall. The upper portion of the liver is overlapped by the right lung (see Fig. 98), and hence at this point we get a certain amount of resonance on percussion, although the tone is not so clear as that to be obtained higher up. Below the sixth rib we find true *flatness* near the sternum and for a few inches to the right of this point. As we go toward the axilla, the line of lung resonance slopes down, as is seen in Fig. 98. In the back resonance extends to the ninth or tenth ribs.

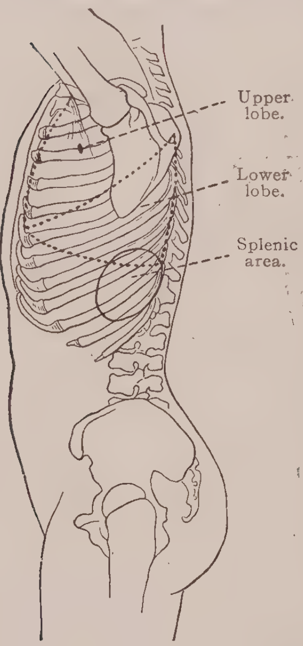

Fig. 99.—Position of the Left Lung in the Axilla.

2. *Normal Dull Areas*

On the left side, the main dull area corresponds to the heart, which at this point approaches the chest wall, and over the portion shaded darkest is uncovered by the lung. The part here lightly shaded corresponds to that portion of the heart which is overlapped by the margin of the right and left lungs.

Over the portion of the heart not overlapped by the lung (see Fig. 98, p. 133) the percussion note is nearly flat to light percussion, and very dull even when strongly percussed. This little quadrangular area is known as the "*superficial cardiac space*," and the dulness corresponding to it is referred to as the "*superficial*" cardiac dulness, while the dulness corresponding to the outlines of the heart itself beneath the overlapping lung margins is called the "*deep*" cardiac dulness.

When the heart becomes enlarged, both of these areas, the deep and the superficial, are enlarged, the former corresponding to the increased size of the heart itself, while the superficial cardiac space is extended because the margins of the lungs are pushed aside and a larger piece of the heart wall comes in contact with the chest wall. Accordingly, either the superficial or the deep dulness may be mapped out as a means of estimating the size of the heart.

The deep cardiac dulness is much more satisfactory as a means of estimating the size of the heart, but much more difficult to map out. The longer that I have tried to percuss the heart area, and the more I have watched others attempt to do so, correlating the results with post-mortem facts and x-ray findings, the less I believe in the value of cardiac percussion. I rarely attempt it.

It is a disputed point whether light or forcible percussion should be used when we attempt to map out the deep cardiac dulness. I find that as years go on I very seldom percuss out any cardiac border except the left and that only in the few cases in which the cardiac apex cannot be palpated. Much too great stress has been laid on percussion of the heart. Palpation and blood-pressure measurements usually give all essential information. When they do not, x-ray is usually the only reliable way to find out the heart's size.

Good observers are to be found on each side of this question, and I have no doubt that cardiac percussion works well in skilled hands. Personally I have found it nearly useless.

Whatever method we use we must percuss successive points along a line running at right angles to the border of the organ which we wish to outline until a change of note is perceived.

If now we look at the upper part of the chest in Fig. 98, we notice at once that the two sides are not shaded alike: the left apex is distinctly lighter colored than the right. This is a very important point and one not sufficiently appreciated by students. The apex of the normal right lung is distinctly less resonant than the apex of the left in a corresponding poition.

In percussing at the bottom of the left axilla, we sometimes find a small oval area of dulness corresponding to that outlined in Fig. 99. This is the area of *splenic dulness*, so called, and corresponds to that portion of the spleen which is in contact with the chest wall. This dull area is to be made out only in case the stomach and colon are not overdistended with air. When these organs are full of gas as is not infrequently the case, there is no area of splenic dulness and the whole region gives forth, when percussed, a note of a quality next to be described, namely, "*tympanitic.*" In my experience, percussion of the spleen is of very little use when we want it most, *i.e.*, in infectious diseases like typhoid, malaria or sepsis. If the edge of the organ can be felt it is almost always enlarged; if the edge cannot be felt the results of percussion are most unreliable. In leucæmia and other diseases which greatly enlarge the spleen we can accurately percuss out its upper border, but this has little practical value.

3. *Tympanitic Resonance*

Tympanitic resonance is that obtained over a hollow body, like the stomach when moderately distended with air.[1] It is usually of a higher pitch than the resonance to be obtained over the normal lung, and may be elicited by percussion lighter than that needed to bring out the lung resonance. It differs also from the vesicular or pulmonary resonance in *quality*, in a way easy to appreciate but difficult to describe. Tympanitic resonance is usually to be heard when one percusses over the front of the left chest near the ensiform cartilage and for a few inches to the left of this point over the area corresponding with that of the stomach bubble as seen by *x*-ray. This tympanitic area, known as "*Traube's semilunar space*," varies a great deal in size according to the contents of the stomach. It is bounded on the right by the liver flatness, above by the pulmonary resonance, on the left by the splenic dulness, and below by the resonance of the intestine, which is also tympanitic, although its pitch is different owing to the different size and shape of the intestine.

(The right axilla shows normal lung resonance down to the point at which the liver flatness begins, as shown in Fig. 98.)

In the back, when the scapulæ are drawn forward, as shown in Fig. 95, page 130, percussion elicits a clear vesicular resonance from top to bottom on each side, although the top of the right lung is always slightly less resonant than the top of the left, and sometimes the bottom of the right lung is slightly less resonant than the corresponding portion of the left.

4. *Krönig's Isthmus*

In percussing the apices behind one can map out on each trapezius muscle a band of resonance bounded by the dulness of the neck structure on the median side and of the dulness of the shoulder structures outwardly. This strip, known as Krönig's isthmus, is narrowed in chronic (usually tuberculous) disease of the lung apex, as lung retraction replaces resonance by dulness.

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[1] *Extreme* distention here, as in a snare drum, is associated with a dull percussion note (see below p 30).

The normally resonant strip 4 to 6 cm. wide may in disease be reduced to 2 or 3 cm.

It should be remembered, however, that in the majority of cases the resonance throughout the back is distinctly less than that obtained over the front, on account of the greater thickness of the back muscles. Yet in children or emaciated persons, or where the muscular development is slight, there may be as much resonance behind as in front.

Importance of Percussing Symmetrical Points.—Since we depend for our standard of resonance upon comparison with a similar spot on the outside of the chest, it is all-important that in making such comparisons we should percuss symmetrical points, and not, for example, compare the resonance over the third rib in the right front with that over the third interspace on the left, since more resonance can always be elicited over an interspace than over a rib. This comparison of symmetrical points, however, is interfered with by the presence of the heart on one side and the liver on the other, as well as by the fact that the apex of the right lung is normally less resonant than that of the left. A resonance which would be pathologically feeble if obtained over the left top may be normal over the right. Where both sides are abnormal, as in bilateral disease of the lung, or where fluid accumulates in both pleural cavities, we have to make the best comparison we can between the sound in the given case and an ideal standard carried in the mind.

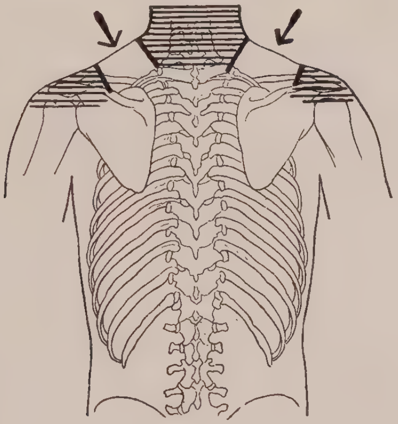

FIG. 100.—The arrows point to Krönig's Isthmus.

It must always be remembered that the amount of resonance obtained at any point by percussion depends upon how hard one strikes, as well as upon the conditions obtaining within the chest. A powerful blow over a diseased lung may bring out more resonance than a lighter blow over a normal lung. To strike with perfect fairness and with equal force upon each side can be learned only by considerable practice. Furthermore, the distance from the ear to each of the two points, the resonance of which we are comparing, must be the same—that is, we must stand squarely in front or squarely behind the patient, otherwise the note coming from the part farther from the ear will sound duller than that coming from the nearer side.

The normal resonance of the different parts of the chest can be considerably modified by the position of the patient, by deep breathing, by muscular exertion, and by other less important conditions. If, for example, the patient lies upon the left side, the heart swings out toward the left axilla and its dulness is extended in the same direction. Deep inspiration distends the margins of the lungs so that they encroach upon and reduce the area of the heart dulness and liver dulness. After muscular exertion the lungs become more than ordinarily voluminous, owing to the temporary distention brought about by the unusual amount of work thrown upon them.

The area of cardiac dulness is increased in any condition involving insufficient lung expansion. Thus, in children, in debility, chlorosis, or fevers, the space occupied by the lungs is relatively small and the dull areas corresponding to the heart and liver are proportionately enlarged. In old age, on the other hand, when the lungs have lost part of their elasticity and sag down over the heart and liver, the percussion dulness of these organs is reduced.

Conditions Modifying the Percussion Note in Health.—The development of muscle or fat as well as the thickness of the chest wall will influence greatly the amount of resonance to be obtained by percussion. Indeed, we see now and then an individual in no part of whose chest can any clear percussion tone be elicited. In women, the amount of development of the breasts has also great influence upon the percussion note. In children, the note is generally clearer, and only the lightest percussion is to be used on account of the thinness of the chest wall. In old people whose chests are almost always more or less barrel-shaped, a shade of tympanitic quality is added to the normal vesicular resonance. Distention of the colon with gas often obliterates the liver dulness by rotating that organ so that only its edge is in contact with the chest wall, and if there is wind in the stomach, a variable amount of tympany is heard on percussing the lower left front and axilla or even in the left back.

If a patient is examined while lying on the side, the amount of resonance over the lung corresponding to the side on which he lies is usually less than that of the side which is uppermost, because there is more air in the latter.[1] Whatever the patient's position, the amount of resonance is also greater at the end of inspiration than at the end of expiration, for the reason just given. As the lungs expand with full inspiration, their borders must move so as to cover a larger portion of the organs which they normally overlap. Portions of the chest which at the end of expiration are dull or flat, owing to the close juxtaposition of the heart, liver, or spleen, become resonant at the end of inspiration. For example, the lower margin of the right lung moves down during inspiration so as to cover a considerably larger portion of the liver.

Percussion as a Means of Ascertaining the Movability of the Lung Borders.—It is sometimes of importance to determine not merely the position of the resting lung but its power to expand freely. This can be ascertained by percussion in the following way: The lower border of the lung resonance, say in the axilla, is carefully marked out. Then percussion is made over a point just *below* the level of the resting lung and at the same time the patient is directed to inspire deeply. If the lung expands and its border moves down, the percussion note will change suddenly from dull to resonant during the inspiration. An excursion of two or three inches can often be demonstrated by this method, which is especially important for the anterior and posterior margins of the lungs. In the axilla Litten's phrenic shadow will give us the same information.

The mobility of the borders of the lung, as determined by this method, is of considerable clinical importance, for an absence of such mobility may indicate pleuritic adhesions. Its amount depends upon various conditions and varies much in different individuals, but complete absence of mobility is always pathological.

5. *Cracked-pot Resonance*

When percussing the chest of a crying child, we sometimes notice that the sound elicited has a peculiar "*chinking*" quality, like that produced by striking one coin with another, but more muffled. The sound may be more closely imitated, and the mode of its production illustrated, by clasping the hands palm to palm so as to enclose an air space which communicates with the outer air through a chink left open, and then striking the back of the under hand against the knee. By the blow, air is forced out through the chink with a sound like that of metallic coins struck together.

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[1] There is also a shade of tympany associated with the dulness of the feebly expanded lung of the lower side.

In disease, the cracked-pot sound is usually produced over a superficial and empty pulmonary cavity (as in advanced phthisis) from which the air is suddenly expelled by the percussion stroke.

It is much easier to hear this peculiar sound if, while percussing, one listens with a stethoscope at the patient's open mouth. The patient himself holds the chest piece of the instrument just in front of his open mouth, leaving the auscultator's hands free for percussing.

6. *Amphoric Resonance*

A low-pitched hollow sound approximating in quality to tympanitic resonance, and occasionally obtained over pulmonary cavities or over pneumothorax, has received the name of amphoric resonance. It may be imitated by percussing the trachea or the cheek when moderately distended with air.

(*a*) *Summary*

The varieties of resonance to be obtained by percussing the norma thorax are:

(1) *Vesicular resonance*, to be obtained over normal lung tissue.

(2) *Tympanitic resonance*, to be obtained in Traube's semilunar space.

(3) *Diminished resonance or dulness*, such as is present over the scapulæ, and

(4) Absence of resonance or *flatness*, such as is discovered when we percuss over the lowest ribs in the right front.

(5) *Cracked-pot resonance*, sometimes obtainable over the chest of a crying child.

(6) *Amphoric resonance*, obtainable over the trachea.

Any of these sounds may denote disease if obtained in portions of the chest where they are not normally found. *Each has its place, and becomes pathological if found elsewhere.*

III. Sense of Resistance

While percussing the chest we must be on the lookout not only for changes in resonance, but for variations in the amount of resistance felt underneath the finger. Normally the elasticity of the chest walls over the upper fronts is considerably greater and the sense of resistance considerably less than that felt over the liver. In the axillæ and over those portions of the back not covered by the scapulæ, we feel in normal chests an elastic resistance when percussing which is in contrast with the dead, woodeny feeling which is communicated to the finger when the air-containing lung is replaced by fluid or solid contents (pleuritic effusion, pneumonia, phthisis, etc.). Muscle spasm over pleurisy or diseased lung may also increase the sense of resistance (Pottenger). In some physicians this sense of resistance is very highly developed and as much information is obtained thereby as through the sounds elicited.

CHAPTER VIII

AUSCULTATION

I. MEDIATE AND IMMEDIATE AUSCULTATION

Auscultation may be practiced by placing one's ear directly against the patient's chest (immediate auscultation) or with the help of a stethoscope (mediate auscultation).

Each method has its place. Immediate auscultation is said to have advantages similar to those of the low power of the microscope, in that it gives us a general idea of the condition of a relatively large area of tissue, while the stethoscope may be used, like the oil immersion lens, to bring out details at one or another point.

On the other hand, I am firmly convinced that the unaided ear can perceive sounds conducted from the interior of the lungs—sounds quite inaudible with any stethoscope—and that in this way the faint tubular breathing produced by deep-seated areas of solidified lung may be recognized.

Immediate auscultation may be objected to:

(*a*) On grounds of *delicacy* (when examining persons of the opposite sex).

(*b*) On grounds of *cleanliness* (although the chest may be covered with a towel so as to protect the auscultator to a certain extent).

(*c*) Because we cannot conveniently reach the supraclavicular or the upper axillary regions in this way.

(*d*) Because it is difficult to localize the different valvular areas and the sites of cardiac murmurs if immediate auscultation is employed.

On account of the latter objection the great majority of observers now use the stethoscope to examine the heart. For the lungs, both methods are employed by many experienced auscultators. I have already mentioned the importance of immediate auscultation in the search for deep-seated areas of pneumonia. Attention has also been called by Conner (Assoc. of American Physicians, 1907, p. 113) to the fact that the diastolic murmur of aortic insufficiency is sometimes audible to the unaided ear when it cannot be heard with any form of stethoscope. Faint, high-pitched blowing sounds are those which the free ear is especially adapted to detect.

This is doubtless due, as Conner explains, to the fact that the tubes of the stethoscope do not conduct high-pitched sounds well. With the free ear we have also the opportunity to detect the bone-conducted sounds which are missed in mediate stethoscopic auscultation.

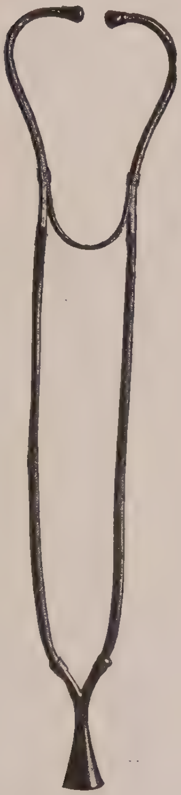

FIG. 101.—Stethoscope Fitted with Long Flexible Tubes, Especially Useful When Examining Children.

II. SELECTION OF A STETHOSCOPE

(1) It is as rash for any one to select a stethoscope without first trying the fit of the ear pieces in his ears as it would be to buy a new hat without trying it on. What suits A. very well is quite impossible for B. It is true that one can get used to almost any stethoscope as one can to almost any hat, but it is not necessary to do so. The ear pieces of the ordinary stethoscope are often too small and rarely too large. In case of doubt, therefore, it is better to err upon the side of getting a stethoscope with too large rather than too small ends.

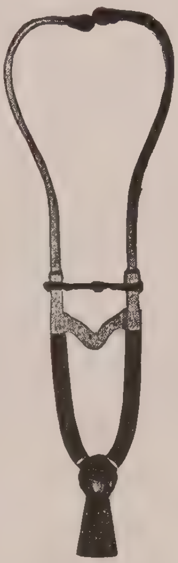

FIG. 102.—Camman Stethoscope with Stiff Tubing and Rubber Strap.

(2) The binaural stethoscope, which is now used almost exclusively in this country, maintains its position in the ears of the auscultator either through the pressure of a rubber strap stretched around the metal tubes leading to the ears, or by means of a steel spring connecting the tubes. Either variety is usually satisfactory, but I prefer a stethoscope made with a steel spring (see Fig. 101) because such a spring is far less likely to break or lose its elasticity than a rubber strap. A rubber strap can always be added if this is desirable. It is important to pick out an instrument possessing a spring not strong enough to cause pain in the external meatus of the ear and yet strong enough to hold the ear pieces firmly in place. Persons with narrow heads need a much more powerful spring or strap than would be convenient for persons with wide heads.

(3) The rubber tubing used to join the metallic tubes to the chest piece of the instrument should be as flexible as possible (see

Fig. 107). Stiff tubing (see Fig. 102) makes it necessary for the auscultator to move his head and body from place to place as the examination of the chest progresses, while if flexible tubing is used the head need seldom be moved and a great deal of time and fatigue is thus saved. Stiff stethoscopes are especially inconvenient when examining the axilla.

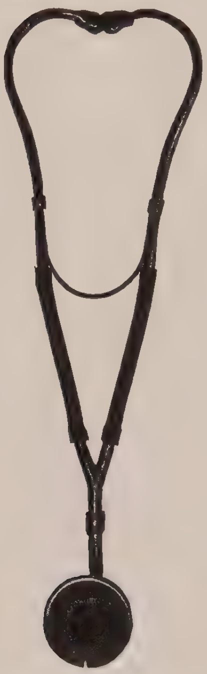

Fig. 103.—Bowles' Stethoscope. Front view.

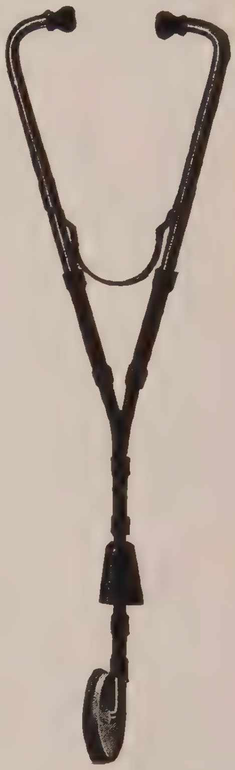

Fig. 104.—Combination Bowles' Stethoscope.

(4) Jointed stethoscopes which fold up or take apart should be scrupulously avoided. They are a delusion and a snare, apt to come apart at critical moments, and to snap and creak at the joints when in use, sometimes producing in this way sounds which may be easily mistaken for râles. Such an instrument is no more portable nor compact than the ordinary form with flexible tubes. It has, therefore, no advantages over stethoscopes made in one piece and possesses disadvantages which are peculiarly annoying.

(5) *The Chest Piece.*—The majority of the stethoscopes now in use have a chest piece of hard-rubber, wood or metal with a diameter of about seven-eighths of an inch. Chest pieces of larger diameter than this are to be avoided as they are very difficult to maintain in close apposition with thin chests. To avoid this difficulty the chest piece is sometimes made of soft-rubber or its diameter still further reduced.

(6) *The Bowles Stethoscope.*—(See Figs. 103 and 104.) Within recent years there has been introduced an instrument which, for many purposes, seems to me far superior to any other form of stethoscope with which I am acquainted. Its peculiarity is the chest piece, which consists of a very shallow steel cup (see Fig. 105) over the mouth of which a thin metal plate or a bit of pigskin is fastened.

The metal or pigskin diaphragm serves simply to prevent the tissues of the chest from projecting into the shallow cup of the chest piece when the latter is pressed against the chest, and does not in any other way contribute to the sounds which we hear with the instrument. This is proved by the fact that we can hear as well even when the diaphragm is cracked across in several directions.

With this instrument almost all sounds produced within the chest can be heard much more distinctly than in any other variety of stethoscope. Cardiac murmurs which are inaudible with any other stethoscope may be distinctly heard with this. Especially is this true of all high-pitched sounds and of the murmurs of aortic regurgitation. Yet it is useful for examination not merely of the heart, but of the lungs as well. For any one who has difficulty in hearing the ordinary cardiac or respiratory sounds, or for one who is partially deaf, the instrument is invaluable. As the metal rim of the chest is apt to get unpleasantly cold, it is best to cover it with a bit of rubber or kid. This saves the patient some discomfort and also tends to prevent the instrument from slipping on the skin. The flat chest piece makes the instrument very useful in listening to the posterior portions of the lungs in cases of pneumonia in which the patient is too sick to be turned over or to sit up. Without moving the patient at all we can work the chest piece in under the back of the patient by pressing down the bed-clothes, and in this way can listen to any part of the chest without moving the patient. A further advantage of the instrument is that it enables us to gain an approximately accurate idea of the heart sounds without undressing the patient. Respira-

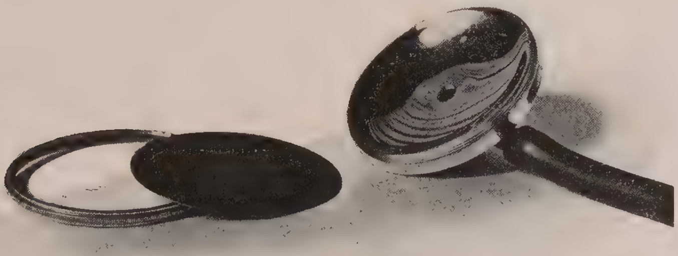

FIG. 105.—Chest Piece of Bowles' Stethoscope. On the right the shallow cup communicating with the ear tubes. On the left the diaphragm which covers the cup, and the ring which holds it in place.

tory sounds cannot well be listened to through the clothes, as the rubbing of the latter may simulate râles.

There are two purposes for which I have sometimes found the Bowles stethoscope inferior to the ordinary stethoscope:

(1) For listening over the apex of the lung for fine râles, *e.g.*, in incipient phthisis.

(2) For listening for *low-pitched or superficial* sounds, such as a friction rub or a presystolic murmur.[1] When I desire to listen for fine râles at an apex, for a friction rub, or for a presystolic murmur, I separate the chest piece of the Bowles stethoscope from the hard-rubber bell into which it is inserted, thereby converting the instrument into one of the ordinary form. With an extra-hard-rubber bell attached, the instrument is no more bulky than an ordinary stethoscope, and far more efficient. When used for listening to the respiration, the Bowles instrument gives us information similar in some respect to that obtained by the use of the free ear—that is we are through it enabled to ascertain by listening at one spot the condition of a much larger area of the chest than can in any other way be investigated.

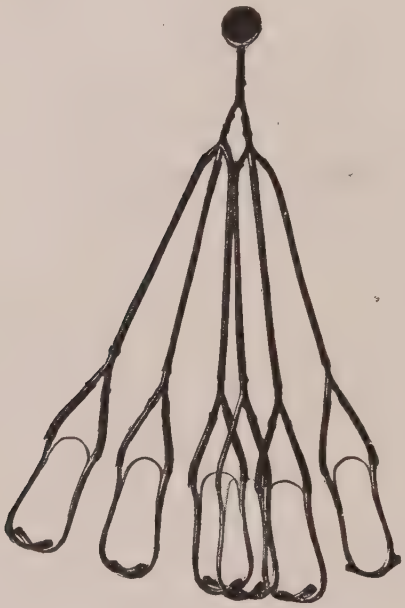

FIG. 106.—Bowles' Multiple Stethoscope for Six Students.

Owing to the fact that both cardiac and respiratory sounds are magnified by the Bowles stethoscope, this instrument is especially well adapted for use with some sort of an attachment whereby several sets of ear pieces are so jointed by tubing to one chest piece that several persons may listen at once. Bowles' multiple stethoscope, fitted for six and for twelve observers, is seen in Figs. 106 and 107, and the method of its use in Fig. 108. In the teaching of auscultation this instrument is of some value, saving as it does the time of the instructor and of the students and the strength of the patient. The sounds conducted through any one of the twelve tubes used in this instrument are as loud as those to be heard with a single instrument of the ordinary form, although far fainter than those to be heard with a single Bowles stethoscope.

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[1] It has frequently been observed, when listening with the ordinary stethoscope, that a presystolic murmur can be better heard if only the very lightest pressure is made with the stethoscope. The fact that a thrill is communicated to the chest wall, and that that thrill is connected with the audible murmur explains my calling this murmur a superficial one.

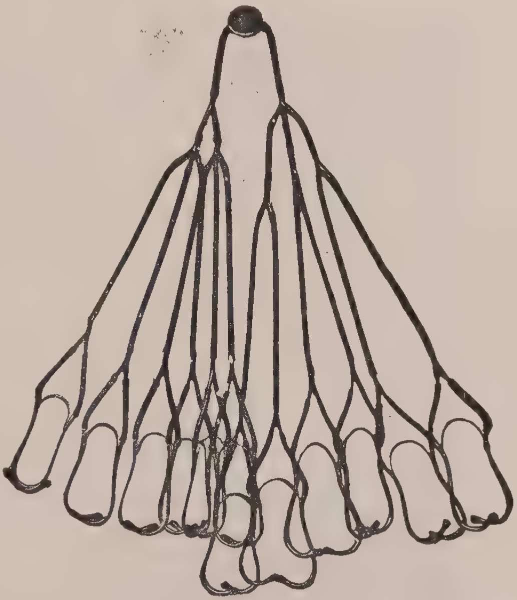
FIG. 107.—Bowles' Multiple Stethoscope for Twelve Students.

III. THE USE OF THE STETHOSCOPE

Having secured an instrument which fits the ears satisfactorily, the beginner may get a good deal of practice by using it upon himself, especially upon his own heart. The chief point to be learned is to *disregard various irrelevant sounds* and to concentrate attention upon those which are relevant. Almost any one hears enough with a stethoscope, and most beginners hear too much. No great keenness of hearing is required, for the sounds which we listen for are not, as a rule, difficult to hear if attention is concentrated upon them.

1. *Selective Attention and What to Disregard*

Accordingly, the art of using a stethoscope successfully depends upon the acquisition of two powers—

(*a*) A knowledge of what to disregard. (*b*) A selective attention or concentration upon those sounds which we know to be of importance.

Among the sounds which we must learn to disregard are the following:

(1) Noises produced in the room or its immediate neighborhood, but not connected with the patient himself. It is, of course, easier to listen in a perfectly quiet room where there are no external noises which need to be excluded from attention, but as the greater part of the student's work must be done in more or less noisy places, it is for the beginner a practical necessity to learn to withdraw his attention from the various sounds which reach his ear from the street, from other parts of the building, or from the room in which he is working. This is at first no easy matter, but can be accomplished with practice.

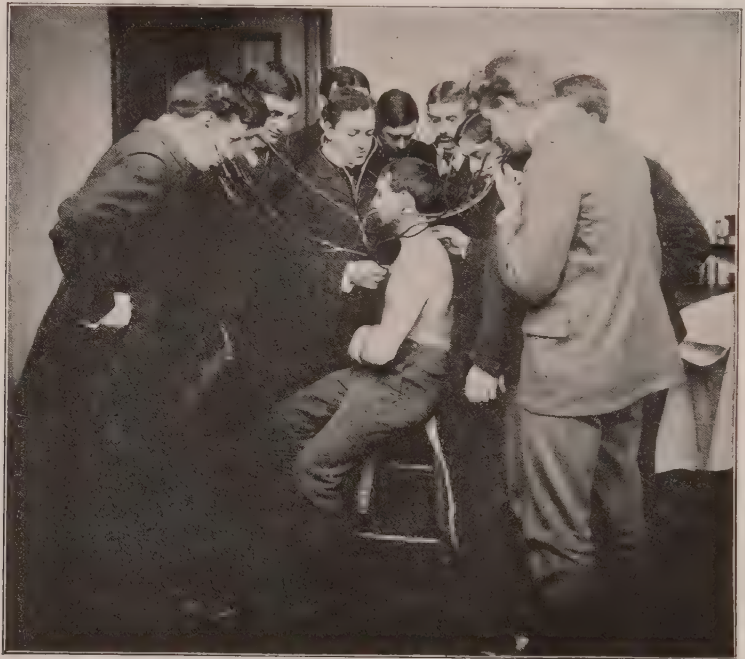

FIG. 108.—Bowles' Multiple Stethoscope in Use. Twelve students listening at once.

(2) When the power to disregard external noises has been acquired, a still further selection must be made among the sounds which come to the ear through the tubes of the stethoscope. Noises produced by friction of the chest piece of the stethoscope upon the skin are especially deceptive and may closely simulate a pleural or pericardial friction sound. It is well for the student to experiment upon the nature and extent of such "skin rubs" by deliberately

moving the chest piece of the stethoscope upon the skin and listening to the sounds so produced. Mistakes can be avoided in the majority of cases by holding the chest piece of the stethoscope very firmly against the chest. This can be easily done when the patient is in the recumbent position, but when the patient is sitting up it may be necessary to press so hard with the chest piece of the stethoscope as to throw the patient off his balance unless he is in some way supported; accordingly, it is my practice in many cases to put the left arm around and behind the patient so as to form a support, against which he can lean when the chest piece of the stethoscope is pressed strongly against his chest. When listening to the back of the chest, the manœuvre is reversed. If the skin is very dry, the ribs are very prominent, or the chest is thickly covered with hair, it may be impossible to prevent the occurrence of adventitious sounds due to friction of the chest piece upon the chest, no matter how firmly the instrument is held. In case of doubt, and in any case in which a diagnosis of fine râles or of pleural or pericardial friction is in question, the chest piece of the stethoscope, the fingers of the hand which holds it and the surface of the chest, at the point where we desire to listen, should be moistened and any hair that may be present thoroughly wetted with a sponge, so that it will lie flat upon the chest. Otherwise the friction of the hair under the chest piece of the stethoscope may simulate crepitant râles as closely as "skin rubs" simulate pleural friction.

(3) The friction of the fingers of the auscultator upon the chest piece or on some other part of the stethoscope frequently gives rise to sounds closely resembling râles of one or another description. The nature of these sounds can be easily learned by intentionally moving the fingers upon the stethoscope. They are to be avoided by wetting the fingers, grasping the bell firmly, and by touching it with as few fingers as will suffice to hold it close against the chest.

(4) Noises produced by a shifting of the parts of the stethoscope upon each other are especially frequent in stethoscopes made in several pieces and jointed together. A variety of snapping and cracking sounds, not at all unlike certain varieties of râles, may thus be produced, and if we are not upon our guard, may lead to errors in diagnosis. Stethoscopes which have no hinges and which do not come apart are far less likely to trouble us in this way.

(5) When a rubber band is used to press the ear pieces more firmly into the ears, a very peculiar sound may be produced by the

breathing of the auscultator as it strikes upon the rubber strap. It is a loud musical note, and may be confused with coarse, dry râles.

When one has learned to recognize and to disregard the noises produced in the ways above indicated, there is still one set of sounds which are very frequently heard, yet which have no significance for physical diagnosis, and must therefore be disregarded; I refer to

2. *Muscle Sounds*

Patients who hold themselves very erect, patients who are chilly or nervous, or who for any reason contract the muscles over which we are listening, produce in these muscles a characteristic set of sounds. The contraction of any muscle in the body produces sounds similar in quality to those heard over the chest, but of less intensity.

Those who have the faculty of contracting the tensor tympani muscle at will can at any time listen to a typical muscle sound. Or close both ears with the fingers and strongly contract the masseter muscle, with the teeth clenched. A low-pitched muscle sound will be heard.

It is well also to have a patient contract one of the pectorals and then listen to the sound thus produced. In some cases a continuous, low-pitched roar or drumming is all that we hear; in other cases we hear nothing but the breath sounds during expiration, while during inspiration the breath sound is obscured by a series of short, dull, rumbling sounds, following each other at the rate of from five to ten in a second. Occasionally the sound is like the puffing of the engine attached to a pile-driver, or like a stream of water falling upon a sheet of metal just slowly enough to be separated into drops and heard at a considerable distance. As already mentioned, we are especially apt to hear these sounds at the end of forced inspiration, owing to the contraction of voluntary muscles during that portion of the respiratory act. They are most often heard over the upper portion of the chest (over the pectorals in front and over the trapezius behind), but in some persons no part of the chest is free from them. It is a curious fact that we are not always able to detect by sight or touch the muscular contractions which give rise to these sounds, and the patient himself may be wholly unaware of them. Under such circumstances they are not infrequently mistaken for râles, and I am inclined to think that many of the sounds recorded as "crumpling," "obscure," "muffled," "distant," or "indeterminate" râles are in reality due to muscular contractions. The adjectives "muffled" and "distant" give us an inkling as to the qualities which distinguish muscular sounds from râles. Râles can often be counted, are more clean cut, have a more distinct beginning and end, seem nearer to the ear, and possess more of a crackling or bubbling quality than muscle sounds.

The joint sounds and fascial rubbings described by F. T. Lord have already been referred to. General relaxation of all muscles usually quiets them.

3. *Other Sources of Error*

Another source of confusion, which for beginners is very troublesome, especially if they are using the ordinary form of stethoscope with a bell-shaped chest piece, arises in case the chest piece is not held perfectly in apposition with the skin. If, for example, the stethoscope is slightly tilted to one side so that the bell is lifted from the skin at some point, or if one endeavors to listen over a very uneven part of the chest on which the bell of the stethoscope cannot be made to rest closely, a roar of external noises reaches the ear through the chink left between the chest piece and the chest. After a little practice one learns instantly to detect this condition of things and so to shift the position of the chest piece that external noises are totally excluded; but by the beginner, the peculiar babel of external noises, which is heard whenever the stethoscope fails to fit closely against the chest, is not easily recognized, and hence he tends to attribute some of these external sounds to diseased conditions within the chest.

Again, it is not until we have had considerable practice that our sense of hearing comes instantly to tell us when something is wrong about the stethoscope itself; when, for example, one of the tubes is blocked, kinked, or disconnected, or when we are holding the stethoscope upside down, so that the ear pieces point downward instead of upward (see Figs. 109 and 110). It is only when we have learned through long practice about how much we ought to hear at a given point in the normal chest that we recognize at once the fact that we are not hearing *as much as we should*, in case some one of the above accidents has happened. Many beginners do not listen long enough in any one place, but move the chest piece of the stethoscope about rapidly from point to point, as they have seen experienced auscultators do; but it is remarkable how much more one can hear at a given point by simply persevering and listening to beat after beat, or breath after breath. It is sometimes difficult to avoid the impression that the sounds themselves have grown louder as we continue to listen, especially if we are in any doubt as to what we hear. Therefore, if we hear indistinctly, it is important to keep on listening, and to fix the attention successively upon each of the different elements in the sounds under consideration. In difficult cases we should use every possible aid toward concentration of the attention, and where it is possible, all sources of distraction should be eliminated. Thus, in any case of doubt, I think it is important for the auscultator to get himself into as comfortable a position as he can, so that his attention is not distracted by his own physical discomforts. Many auscultators shut their eyes when listening in a difficult case so as to avoid the distraction of impressions coming through the sense of sight. It goes without saying that if quiet can be secured in the room where we are working, and outside it as well, we shall be enabled to listen much more profitably. Hawes[1] has studied the sources of error in auscultation with special reference to the diagnosis of tuberculosis. He gives the following figures: In 250 cases muscle sounds were marked in 57, slight in 25, absent in 168. Joint sounds were present in 126 of 250, tendon or bursal sounds in 10 cases, atelectatic crackles in 5 cases. In 55 instances, or 22 per cent., the joint sounds resembled râles.

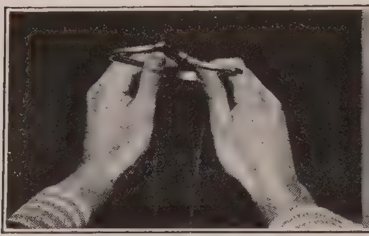

FIG. 109.—Stethoscope Held Right Side Up.

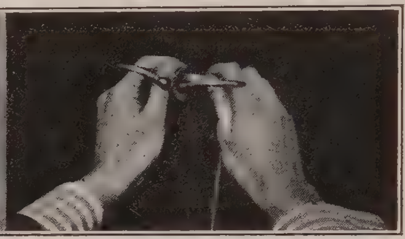

FIG. 110.—Stethoscope Held Wrong Side Up.

IV. AUSCULTATION OF THE LUNGS

In the majority of cases ordinary quiet breathing is not forcible enough to bring out the sounds on which we depend for the diagnosis of the condition of the lungs. Deep or forced breathing is what we need.

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[1] Hawes: *Boston Med. and Surg. Journ.*, June 18, 1914.

As a rule, the patient must be taught how to breathe deeply, which is best accomplished by personally demonstrating the act of deep breathing and then asking him to do the same. Two difficulties are encountered:

(*a*) The patient may blow out his breath forcibly and with a noise, since that is what he is used to doing whenever he takes a long breath under ordinary circumstances; or

(*b*) It may be that he cannot be made to take a deep breath at all. The first of these mistakes alters the sounds to be heard with the stethoscope in any part of the chest by disturbing both the rhythm and the pitch of the respiratory sounds. In this way the breathing may be made to sound tubular or asthmatic throughout a sound chest. This difficulty can sometimes be overcome by demonstrating to the patient that what you desire is to have him open his mouth, take a full breath and then simple *let it go*, but *not* blow it forcibly out. In some cases the patient cannot be taught this, and we have to get on the best we can despite his mistakes. When he cannot be made to take a full breath at all, we can often accomplish the desired result by getting him to cough. The breath just before and after a cough is often of the type we desire. The use of voluntary cough in order to bring out râles will be discussed later on. Another useful manœuvre is to make the patient count aloud as long as he can with a single breath. The deep inspiration which he is forced to take after this task is of the type which we desire.

1. *Respiratory Types*

In the normal chest two types of breathing are to be heard:

(1) Tracheal, bronchial, or tubular breathing.
(2) Vesicular breathing.

Tracheal, bronchial, or tubular breathing is to be heard in normal cases if the stethoscope is pressed against the trachea, and as a rule it can also be heard over the primary bronchi, in front or behind (see Figs. 111 and 112).

Vesicular breathing is to be heard over most of the remaining portions of the lung—that is, in the front of the thorax except where the heart and the liver come against the chest wall, in the back except over the scapulæ, and in the axillæ. Two exceptions to this statement will be mentioned later.

(a) *Characteristics of Vesicular Breathing*

Vesicular breathing—that heard over the air vesicles or parenchyma of the lung—has certain characteristics which I shall try to describe in terms of intensity, duration, and pitch.

Of the *quality* of the sounds heard over this portion of the lung there is little can be said; it sounds something like the swish of the wind in a grove of trees some distance off, and hence is sometimes spoken of as "breezy."

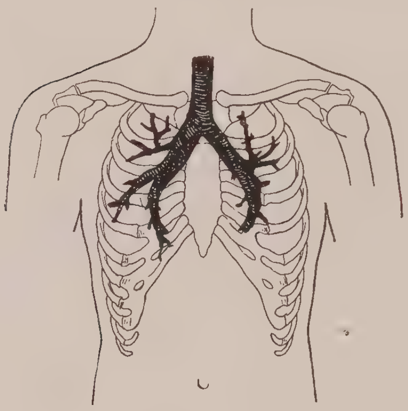

Fig. 111.—Position of Trachea and Bronchi from the Front.

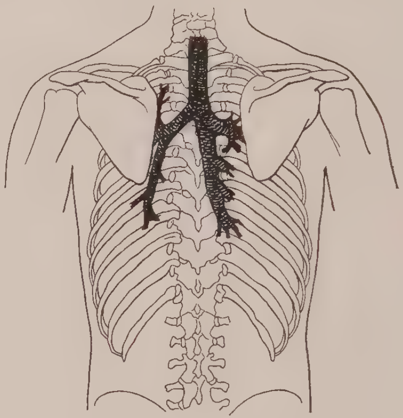

Fig. 112.—Position of Trachea and Bronchi from the Back.

The intensity, duration, and pitch of the inspiration as compared with that of the expiration may be represented as in Fig. 113. In this figure, as in all those to be used in description of respiratory sounds—

(1) I represent the inspiration by an up-stroke and the expiration by a down-stroke (see the direction of the arrows in Fig. 113).

(2) The *length* of the up-stroke as compared with that of the down-stroke corresponds to the *length* of inspiration compared with expiration.

(3) The *thickness* of the up-stroke as compared with the down-stroke represents the *intensity* of the inspiration as compared with the expiration.

(4) The *pitch* of inspiration as compared with that of expiration is represented by the *sharpness of the angle* which the up-stroke makes with the perpendicular as compared with that which the down-stroke

makes with the perpendicular. The *pitch of a roof* may be thought of in this connection to remind us of the meaning of these symbols.

If now we look again at Fig. 113 we see that when compared with expiration (the down-stroke), the inspiration is—

(*a*) More intense.
(*b*) Longer.
(*c*) Higher pitched.

Our comparison is invariably made between inspiration and expiration, and not with any other sound as a standard.

Now, this type of breathing (which, as I have said, is to be heard over every portion of the lung except those portions immediately adjacent to the primary bronchi), is not heard everywhere with equal intensity. It is best heard below the clavicles in front, in the axillæ, and below the scapulæ behind; over the thin, lower edges of the lung, whether behind or at the sides, it is feebler, though still retaining its characteristic type as revealed in the inspiration and expiration in respect to intensity, duration, and pitch. To represent distant vesicular breathing graphically we have only to draw its symbol on a smaller scale (see Fig. 114). On the other hand, when one listens to the lungs of a person who has been exerting himself strongly, one hears the same type of respiration, but on a *larger scale*, which may then be represented as in Fig. 115. This last symbol may also be used to represent the respiration which we hear over normal but thin-walled chests; for example, in children or in emaciated persons. It is sometimes known as "*exaggerated*" or "*puerile*" respiration. When one lung is thrown out of use by disease so that increased work is brought upon the other, the breath sounds heard over the latter are increased and seem to be produced on a larger scale. Such breathing is sometimes spoken of as "rough" breathing.

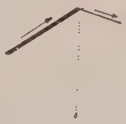

FIG. 113.—Vesicular Breathing.

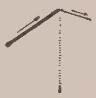

FIG. 114.—Distant Vesicular Breathing.

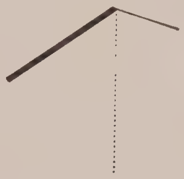

FIG. 115.—Exaggerated Vesicular Breathing.

It is very important to distinguish at the outset between the different *types* of breathing, one of which I have just described, and the different *degrees* of loudness with which any one type of breathing may be heard.

(*b*) *Bronchial or Tracheal Breathing in Health*

Bronchial breathing may be symbolically represented as in Fig. 116, in which the increased length of the down-stroke corresponds to the increased duration of expiration, and the greater thickness of both lines corresponds to the greater intensity of both sounds, expiratory and inspiratory, while the sharp pitch of the "*gable*" on both sides of the perpendicular corresponds to the high pitch of both sounds. Expiration, in most cases, slightly exceeds inspiration both in intensity and pitch, and considerably exceeds it in duration, while as compared with vesicular breathing almost all the relations are reversed. Bronchial breathing has also a peculiar quality which can be better appreciated than described.

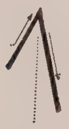

FIG. 116.—Bronchial Breathing of Moderate Intensity.

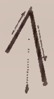

FIG. 117.—Distant Bronchial Breathing.

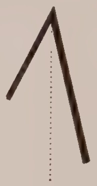

FIG. 118.—Very Loud Bronchial Breathing.

In the healthy chest this type of breathing is to be heard if one listens over the trachea or primary bronchi (see above, Fig. 111), but practically, one hardly ever listens over the trachea and bronchi except by mistake, and the importance of familiarizing one's self with the type of respiration heard over these portions of the chest is due to the fact that in certain diseases, especially in pneumonia and phthisis, we may hear bronchial breathing over the *parenchyma* of the lung where normally vesicular breathing should be heard.

The student should familiarize himself with each of these types of breathing, the vesicular and the bronchial, concentrating his attention as he listens first upon the inspiration and then upon the expiration, and comparing them with each other, first in duration, next in intensity, and lastly in pitch. To those who have not a musical ear, high-pitched sounds convey the general impression of being shrill, while low-pitched sounds sound hollow and empty, but the distinction between intensity and pitch is one comparatively difficult to master. Distant bronchial breathing may be represented in Fig. 116, and is to be heard over the back of the neck opposite the position of the trachea and bronchi. Fig. 118 represents very loud bronchial breathing such as is sometimes heard in pneumonia.

(c) *Broncho-Vesicular Breathing in Health*

As indicated by its name, this type of breathing is intermediate between the two just described, hence the terms "mixed breathing," or "atypical breathing" ("unbestimmt"). Its characteristics may be symbolized as in Fig. 119. In the normal chest one can become familiar with broncho-vesicular breathing, by examining the apex of the right lung, or by listening over the trachea or one of the primary bronchi, and then moving the stethoscope half an inch at a time toward one of the nipples. In the course of this journey one passes over points at which the breathing has, in varying degrees, the characteristics intermediate between the bronchial type from which we started and the vesicular type toward which we are moving. Expiration is a little longer, intenser, or higher pitched than in vesicular breathing, and inspiration a little shorter, feebler, or lower pitched; but since these characteristics are variously combined, there are many subvarieties of broncho-vesicular breathing which, for purposes of convenience (see below, page 300), I have called the *first type of broncho-vesicular breathing* (see Figs. 119, *a*, and 120, *a*) and the *second type of broncho-vesicular breathing* (Figs. 119, *b*, and 120, *b*) or *B–V–I*, and *B–V–II*. The first type is identical with that often called "sharp"—because inspiration is sharp or high pitched. In this type the inspiration is often segmented ("cog-wheel breathing").

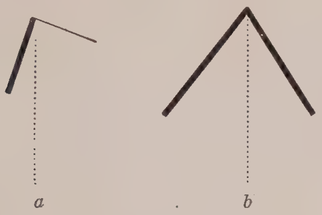

FIG. 119.—Two Common Types of Broncho-Vesicular Breathing.

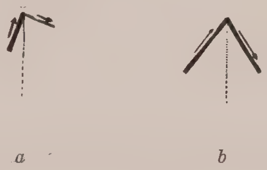

FIG. 120.—Distant Broncho-Vesicular Breathing.

(e) *The Breathing in Asthma*

In this type of breathing, both sounds are usually obscured to a great extent by the presence of piping and squeaking râles (see below).

(f) *Interrupted or "Cogwheel" Breathing*

As a rule, only the inspiration is interrupted, being transformed into a series of short, irregular, jerky puffs as shown in Fig. 123.

Very rarely the expiration is also divided into segments. Inspiration is also abnormally high pitched in most cases. When heard over the entire chest, cogwheel breathing is usually the result of nervousness, fatigue, or chilliness on the patient's part. With the removal of these causes, this type of respiration is confined to a relatively small portion of the chest, and remains present despite the exclusion of fatigue, nervousness, or cold. It points to a local catarrh in the finer bronchi such as to render difficult the entrance of air into the alveoli. As such, it has a certain significance in the diagnosis of early phthisis, a significance similar to that of râles or other signs of localized bronchitis (see below). Cogwheel breathing must be distinguished from cardio-respiratory murmurs which have the qualities of breath sounds, *but occur simultaneously with each cardiac systole.* Such murmurs are very often heard at the left base behind, but have, so far as I know, no clinical importance.

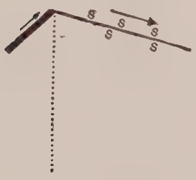

FIG. 121.—Asthmatic Breathing. *s, s, s,* squeaking (musical) râles.

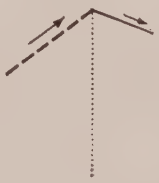

FIG. 122.—Cogwheel Breathing.

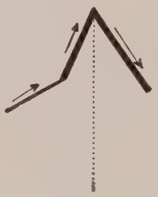

FIG. 123.—Metamorphosing Breathing.

(*g*) *Amphoric or Cavernous Breathing* (see below, p. 162).

(*h*) *Metamorphosing Breathing*

Occasionally, while we are listening to an inspiration of normal pitch, intensity, and quality, a sudden metamorphosis occurs and the type of breathing changes from vesicular to bronchial or amphoric (see Fig. 123), or the intensity of the breath sounds may suddenly be increased without other change. These metamorphoses are usually owing to the fact that a plugged bronchus is suddenly opened by the force of the inspired air, so that the sounds conducted through it become audible.

V. DIFFERENCES BETWEEN THE TWO SIDES OF THE CHEST

(*a*) Over the apex of the right lung—that is, above the right clavicle in front, and above the spine of the scapula behind—one hears in the great majority of normal chests a distinctly broncho-vesicular type of breathing. In a smaller number of cases this same type of breathing may be heard just below the right clavicle. These facts cannot be too strongly insisted upon, since it is only by bearing them in mind that we can avoid the mistake of diagnosing a beginning consolidation of the right apex where none exists. *Breath sounds which are perfectly normal over the right apex would mean serious disease if heard over similar portions of the left lung.* It will be remembered that the apex of the right lung is also duller on percussion than the corresponding portion of the left, and that the voice sounds and tactile fremitus are normally more intense on the right (see Fig. 86). The best explanation of these differences seems to me that given by Petterolf (Archives of Int. Med., Feb., 1909), who has shown that the apex of the right lung is in close contact with the trachea, while the left lung-apex is separated from the trachea by the large blood vessels, the gullet and other structures. The tracheal or bronchial sounds are therefore better transmitted to the right lung.

(*b*) At the base of the left lung, posteriorly, one often hears a slightly rougher or more noisy type of breathing than at the right base. The vicinity of the stomach bubble may explain this.[1]

VI. Pathological Modifications of Vesicular Breathing

Having now distinguished the different types of breathing and described their distribution in the normal chest, we must return to the normal or vesicular breathing in order to enumerate certain of its modifications which are important in diagnosis.

1. *Exaggerated Vesicular Breathing* ("*Compensatory*" *Breathing*)

(*a*) It has already been mentioned that in children or in adults with very thin and flexible chests the normal breath sounds are heard with relatively great distinctness; also that after any exertion which leads to abnormally deep and forcible breathing a similar increase in the intensity of the respiratory sounds naturally occurs.

(*b*) The term "*compensatory breathing,*" or "vicarious" breathing, refers to vesicular breathing of an exaggerated type, such as is heard, for example, over the whole of one lung when the other lung is thrown out of use by the pressure of an accumulation of air or fluid in the pleural cavity. A similar exaggeration of the breathing upon the sound side takes place when the other lung is solidified, as by tuberculosis, pneumonia, or malignant disease, or when it is compressed by the adhesions following pleuritic effusion, or by a contraction of the bones of that side of the chest such as occurs in spinal curvature.

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[1] If the patient lies on the side, that side shows a slightly more tubular respiration with increased voice, whisper, and fremitus. This must be allowed for in all comparisons made in this position.

2. *Diminished Vesicular Breathing*

The causes of a diminution in the intensity of the breath sounds, without any change in their type, are very numerous. I shall mention them in an order corresponding as nearly as possible to the relative frequency of their occurrence.

(*a*) *Fluid, Air or Solid in the Pleural Cavity.*—Probably the commonest cause for a diminution or total abolition of normal breath sounds is an accumulation of fluid in the pleural cavity such as occurs in inflammation of the pleura or by transudation (hydrothorax). In such cases the layer of fluid intervening between the lung and the stethoscope of the auscultator causes retraction of the lung so that little or no vesicular murmur is produced in it, and hence none is transmitted to the ear of the auscultator. An accumulation of air in the pleural cavity (pneumothorax) may diminish or abolish the breath sounds precisely as a layer of fluid does; in a somewhat different way a thickening of the costal or pulmonary pleura or a malignant growth of the chest wall may render the breath sounds feeble or prevent their being heard, because the vibrations of the thoracic sounding-board are thus deadened. Whichever of these causes, fluid or air or solid, intervenes between the lung and the ear of the auscultator, the breath sounds are deadened or diminished without, as a rule, any modification of their type. The amount of such diminution depends roughly on the thickness of the layer of extraneous substance, whether fluid, air, or solid.

Total absence of breath sounds may therefore be due to any one of these causes, provided the layer intervening between the lung and chest wall is of sufficient thickness to produce complete atelectasis of the lung or to deaden the vibrations of the chest wall.

(*b*) In the earliest stages of *pneumonia* the breathing may be feeble or nearly suppressed, not bronchial. In *bronchitis* the breath sounds are sometimes considerably diminished owing to the filling up of the bronchi with secretion. This diminution, however, usually attracts but little attention, owing to the fact that the bubbling and squeaking sounds, which result from the passage of air through the bronchial secretions, distract our notice to such an extent that we find it difficult to concentrate attention upon the breath sounds, even if we do not forget altogether to listen to them. When, however, we succeed in listening *through the râles* to the breath sounds themselves, we usually notice that they are very feeble, especially over the lower two-thirds of the chest. *Œdema of the lung* may diminish the breath sounds in a similar way.

(*c*) *Pain in the thorax*, such as is produced by dry pleurisy or intercostal neuralgia, diminishes the breath sounds because it leads the patient to restrain, so far as possible, the movements of his chest, and so of his lungs. If, for any other reason, the full expansion of the lung does not take place, whether on account of the feebleness of the respiratory movements or because the lung is mechanically hindered by the presence of pleuritic adhesions, the breath sounds are proportionately feeble.

(*d*) *Occlusion of the upper air passages*, as by spasm or œdema of the glottis, renders the breathing very feeble on both sides of the chest. If one of the primary bronchi is occluded, or narrowed as the result of a gumma or a tumor, or by an enlarged gland from without, we may get a unilateral enfeeblement of the breathing.

(*e*) Atelecctasis, especially in new-born infants, gives feeble or absent breathing over the portion affected. Occasionally a paralysis of the muscles of respiration on one or both sides is found to result in a unilateral or bilateral enfeeblement of the breathing.

(*f*) *Relaxation of the lung above a high diaphragm*, itself raised by abdominal tumors, gaseous or fluid accumulations, often produces an area of dulness and feeble breathing with more or less broncho-vesicular quality. *X*-ray helps us very much in recognizing this condition.

One should remember, when estimating the intensity of the breathing, that the sounds heard over the right base are, normally, feebler than those over the left base.

VII. Bronchial or Tubular Breathing in Disease

(*a*) I have already described the occurrence of bronchial breathing in parts of the normal chest, namely, over the trachea and primary bronchi. In disease, bronchial breathing may be heard elsewhere in the chest and usually points to solidification of that portion of lung from which it is conducted. It is heard most commonly in *phthisis* (see below, p. 285).

(*b*) *Croupous pneumonia* is probably the next most frequent cause of bronchial breathing, although by no means every case of croupous pneumonia shows this sign. For a more detailed account of the conditions under which it does or does not occur in croupous pneumonia, see below, p. 287. Lobular pneumonia is rarely manifested by tubular breathing.

(*c*) In about one-third of the cases of pleuritic effusion, distant bronchial breathing is to be heard over the fluid. On account of the feebleness of the breath sounds in such cases they are often put down as absent, as we are so accustomed to associate intensity with the bronchial type of breathing. One should be always on the watch for any degree of intensity of bronchial breathing from the feeblest to the most distinct. In empyema—especially in children—the bronchial breathing heard over the fluid may be intense and often leads to a false diagnosis of unresolved pneumonia or phthisis.

When the breath sounds are enfeebled at the base of the thorax by accumulation of fluid there is usually a layer of bronchial or broncho-vesicular breathing a little higher up near the root of the lung which is relaxed above the fluid. (See also changes associated with pericardial effusion, p. 239.)

(*d*) Rarer causes of bronchial breathing are pleural thickening, hemorrhagic infarction of the lung, syphilis, or malignant disease, any one of which may cause a solidification of a portion of the lung.

VIII. Broncho-vesicular Breathing in Disease

Respiration of this type should be carefully distinguished from puerile or exaggerated breathing, in which we hear the normal vesicular respiration upon a large scale. I have already mentioned that broncho-vesicular breathing is normally to be heard over the apex of the right lung. In disease, broncho-vesicular breathing is heard in other portions of the lung, and usually denotes a moderate degree of solidification of the lung, such as occurs in early phthisis or in the earliest and latest stages of croupous pheumonia. In cases of pleuritic effusion, one can usually hear broncho-vesicular breathing over the upper portion of the affected side, owing to the retraction of the lung at that point.

IX. Amphoric Breathing (*Amphora = A Jar*)

Respiration having a hollow, empty sound like that made by blowing across the top of a bottle, is occasionaly heard in disease over pulmonary cavities (*e.g.*, in phthisis) or in pneumothorax, *i.e.*, under conditions in which the air passes in and out of a large empty cavity within the chest. Amphoric breathing never occurs in health. The pitch of both sounds is low, *but that of expiration lower than that of inspiration.* The intensity and duration of the sounds vary, and the distinguishing mark is their quality which resembles that of a whispered "*who.*"

X. RÂLES

The term "râles" is applied to sounds produced by the passage of air through bronchi which contain mucus or pus, or which are narrowed by swelling of their walls.[1] Râles are best classified as follows:

(1) Crackling râles, coarse or fine. When these are loud or sharp we call them consonating.

(2) Musical râles (squeaks or groans).

Each of these varieties will now be described more in detail.

1. *Coarse Crackles* (*or Bubbles*)

The nature of these is sufficiently indicated by their name. The coarsest or largest bubbles are those produced in the trachea, and ordinarily known as the "*death rattle.*" Tracheal râles occur in any condition involving either profound unconsciousness or very great weakness, so that the secretions which accumulate in the trachea are not coughed out. Tracheal râles are by no means a sure precursor of death, although they are very common in the moribund state. They can usually be heard at some distance from the patient and without a stethoscope. In catarrh of the smaller bronchi, large bubbling râles are occasionally to be heard. In phthisical cavities one sometimes hears coarse, bubbling râles of a very metallic and gurgling quality (see below, p. 302). The finer grades of râles correspond to the finer bronchi.

In the majority of cases these râles are most numerous during inspiration and especially during the latter part of this act. Their relation to respiration may be represented graphically as in Fig. 124, using large dots for coarse râles and small dots for fine râles. Musical râles can be symbolized by the letter *S* (squeaks).

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[1] Râles are of all auscultatory phenomena the easiest to appreciate, provided we exclude various accidental sounds which may be transmitted to the ear as a result of friction of the stethoscope against the skin or against the fingers of the observer. (See above, page 144.)

2. *Fine Crackling Râles*

These differ from the preceding variety merely by the absence of any distinct bubbling quality. They are usually to be heard in cases of bronchopneumonia (tuberculous or not) and in other diseases causing pulmonary condensation so that the tiny explosions of fluid in the bronchi resonate or consonate in the surrounding solid patches. They are especially apt to come at the end of inspiration, a large number being evolved in a very short space of time, so that one often speaks of an "*explosion* of fine crackling râles" at the end of inspiration. There is some doubt whether or not fine crackles can be produced in a pleural exudate, old or new, but personally I am convinced that they *are* not infrequently so produced.

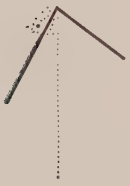

FIG. 124.—Explosion of Fine Râles at End of Inspiration.

The finest sounds of this type are very much like the noise which is heard when one takes a lock of hair between the thumb and first finger, and rubs the hairs upon each other while holding them close to the ear. A very large number of minute crackling sounds is heard following each other in rapid succession. To the inexperienced ear they may seem to blend into a continuous sound, but with practice the component parts may be distinguished. This type of râles is especially apt to occur during inspiration alone, but not very infrequently they are heard during expiration as well.

They are oftenest heard at the end of a case of pneumonia, less frequently at its beginning. In tuberculosis, infarction, pulmonary abscess or œdema they are occasionally heard. If the chest is covered with hair, sounds precisely like crepitant râles may be heard when the stethoscope is placed upon the hairy portions. To avoid mistaking these sounds for râles one must thoroughly wet the hair.

(*a*) *Crackling Râles in Atelectasis*

Fine crackles are very often to be heard along the thin margins of the lungs at the base of the axillæ and in the back, especially when a middle-aged or elderly patient who is breathing superficially first takes deep breaths. In such cases, they usually disappear after the first few respirations, and are then to be explained by the tearing apart of the slightly agglutinated surfaces of the finer bronchioles.

It is by no means invariably the case, however, that such crepitant râles are merely transitory in their occurrence. In a small number of cases they persist despite deep breathing. The frequency of crepitant râles, persistent or transitory, heard over the inferior margin of the normal lung at the bottom of the axilla, is shown by the following figures: Out of 356 normal chests to which I have listened especially for these râles, I found 228, or 61 per cent., which showed them on one or both sides. They are very rarely to be heard in persons under twenty years of age. After forty-five, on the other hand, it is unusual *not* to find them. In my experience they are considerably more frequent in the situation shown in Fig. 182 than in any other part of the lung, but they may be occasionally heard in the back or elsewhere. In view of these facts, it seems to me that we must recognize that it is almost if not quite physiological to find the finer varieties of crackling râles at the base of the axillæ in persons over forty years old. I have supposed these râles to be due to a partial atelectasis resulting from disuse of the thin lower margin of the lungs. Such portions of the lung are ordinarily not expanded unless the respirations are forced and deep.[1]

In certain cases of dry pleurisy there occur fine crackling sounds which can scarcely be differentiated from crepitant râles. I shall return to the description of them, in speaking of pleural friction (see below, p. 328).

3. *Musical Râles* (*Squeaks and Groans*)

The passage of air through moist bronchial tubes narrowed by inflammatory swelling of their lining membrane (bronchitis), by dropsical effusions or by spasmodic contraction (asthma), gives rise not infrequently to a multitude of musical sounds. Such a stenosis occurring in relatively large bronchial tubes produces a deep-toned *groaning* sound, while narrowing of the finer tubes results in *piping*, *squeaking*, *whistling* noises of various qualities. Such sounds are often known as "*dry râles*" in contradistinction to those above described, but as many non-musical crackling râles have also a very dry sound, it seems to me best to apply the more distinctive term "*musical râles*" to all adventitious sounds of distinctly musical quality, giving up the term "dry" altogether. Musical râles are of all adventitious sounds the easiest to recognize but also the most *fugitive and changeable.* They appear now here, now there, shifting from minute to minute, and may totally disappear from the chest and reappear again within a very short time. This is to some extent true of all varieties of râles, but especially of the squeaking and groaning varieties. A cough may elicit them.

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[1] So as to expand the lung and produce the "entfaltungsgeräusch" of the Germans.

Musical râles are heard, as a rule, more distinctly during expiration, especially when they occur in connection with asthma. In these diseases one may hear quite complicated chords from the combinations of râles which vary in pitch.

XI. The Effects of Cough

The influence of coughing upon râles may be either to intensify them and bring them out where they have not previously been heard, or to clear them away altogether. Lying on the side multiplies and intensifies râles on the lower side. Other effects of coughing upon physical signs will be mentioned later.

XII. Pleural Friction

The surfaces of the healthy pleural cavity are lubricated with sufficient serum to make them pass noiselessly over each other during the movements of respiration. But when the tissues become abnormally dry, as in Asiatic cholera, uræmia, or when the serous surfaces are roughened by the presence of a fibrinous exudation, as in ordinary pleurisy, the rubbing of the two pleural surfaces against one another produces peculiar and very characteristic sounds known as "*pleural friction sounds*." The favorite seat of pleural friction sounds is at the bottom of the axilla, *i.e.*, where the lung makes the widest excursion and where the costal and diaphragmatic pleura are in close apposition (see Fig. 82). In some cases pleural friction sounds are to be heard altogether below the level of the lung. In others they may extend up several inches above its lower margin, and occasionally it happens that friction may be appreciated over the whole lung from the top to the bottom. Sometimes friction sounds are heard only at the apex of the lung in early tuberculosis.

The sound of pleural friction may be closely imitated by holding the thumb and forefinger close to the ear, and rubbing them past each other with strong pressure, or by pressing the palm of one hand over the ear and rubbing upon the back of this hand with the fingers of the other. Pleural friction is usually a catchy, jerky, interrupted, irregular sound, and is apt to occur during inspiration only, and particularly at the end of this act. It may, however, be heard with both respiratory acts, but rarely, if ever, occurs during expiration alone.

The intensity and quality of the sounds vary a great deal, so that they may be compared to *grazing*, *rubbing*, *rasping*, and *creaking* sounds. They are sometimes spoken of as "*leathery*." As a rule, they seem very near to the ear, and are sometimes startlingly loud. In many cases they cannot be heard after the patient has taken a few full breaths, probably because the rough pleural surfaces are smoothed down temporarily by the friction which deep breathing produces. After a short rest, however, and a period of superficial breathing, pleural friction sounds often return and can be heard for a short time with all their former intensity. They are increased by pressure exerted upon the outside of the chest wall. Such pressure had best be made with the hand or with the Bowles stethoscope, since the sharp edges of the chest-piece of the ordinary stethoscope may give rise to considerable pain; but if such pressure is made with the hand, one must be careful not to let the hand shift its position upon the skin, else rubbing sounds may thus be produced which perfectly simulate pleural friction. In well-marked cases friction can be *felt* if the hand is laid over the suspected area; occasionally the vibrations are so coarse that they can be heard and felt by the patient himself or by those around him. I have already mentioned F. T. Lord's[1] account of a sound a good deal like pleural friction, often heard over the scapulæ when examining patients whose arms are folded across the chest with each hand on the opposite shoulder. The sound apparently starts in the shoulder-joint on one side or both sides—usually both. It is less jerky and irregular than pleural friction, can often be abolished by shifting the position of the arms, and causes no pain.

XIII. Auscultation of the Spoken or Whispered Voice Sounds

The more important of these is:

1. *The Whispered Voice*

The patient is directed to whisper "one, two, three," or "ninety-nine," while the auscultator listens over different portions of the chest to see to what degree the whispered syllables are transmitted. In the great majority of normal chests the whispered voice is to be heard only over the trachea and primary bronchi in front and behind, while over the remaining portions of the lung little or no sound is to be heard. When, on the other hand, solidification of the lung is present, the whispered voice may be distinctly heard over portions of the lung relatively distant from the trachea and bronchi; for example, over the lower lobes of the lung behind. The usefulness of the whispered voice in the search for small areas of solidification or for the boundaries of a solidified area is great, especially in pneumonia when we wish to save the patient the pain and fatigue of taking deep breaths. Whispered voice sounds are practically equivalent to a forced expiration and can be obtained with very little exertion on the patient's part. The increased transmission of the whispered voice is, in my opinion, a more delicate test for solidification than tubular breathing. The latter sign is present only when a considerable area of lung tissue is solidified, while the increase of the whispered voice may be obtained over much smaller areas. Retraction of the lung above the level of a pleural effusion causes a moderate increase in the transmission of the whispered voice, and at times this increased or bronchial whisper is to be heard over the fluid itself, probably by transmission from the compressed lung above.

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[1] F. T. Lord: Boston Med. & Surg. Journal, Oct. 21, 1909.

Where the lung is completely solidified the whispered words may be clearly distinguished over the affected area. In lesser degrees of solidification the syllables are more or less blurred.

2. *The Spoken Voice*

The evidence given us by listening for the spoken voice in various parts of the chest is considerably less in value than that obtained through the whispered voice. As a rule, it corresponds with the tactile fremitus, being increased in intensity by the same causes which increase tactile fremitus, viz., solidification or condensation of the lung, and decreased by the same causes which decrease tactile fremitus—namely, by the presence of air or water in the pleural cavity, by the thickening of the pleura itself, or by an obstruction of the bronchus leading to the part over which we are listening. In some cases the presence of solidification of the lung gives rise not merely to an increase in transmission of the spoken voice, but to a change in its quality, so that it sounds abnormally concentrated, nasal, and near to the listener's ear. The latter change may be heard over areas where tactile fremitus is not increased, and even where it is diminished. Where this change in the quality of the voice occurs, the actual words spoken can often be distinguished in a way not usually possible over either normal or solidified lung. "Bronchophony," or the distinct transmission of audible words, and not merely of diffuse, unrecognizable voice sounds, is considerably commoner in the solidifications due to pneumonia than in those due to phthisis; it occurs in some cases of pneumothorax and pulmonary cavity.

3. *Egophony*

Among the least important of the classical physical signs is a nasal or squeaky quality of the sounds which reach the observer's ear when the patient speaks in a natural voice. To this peculiar quality of voice the name of "egophony" has been given. It is most frequently heard in cases of moderate-sized pleuritic effusion just about the level of the lower angle of the scapula and in the vicinity of that point. Less often it is heard at the same level in front. It is very rarely heard in the upper portion of the chest and is by no means constant either in pleuritic effusion or in any other condition. A point at which it is heard corresponds not, as a rule, with the upper level of the accumulated fluid, as has been frequently supposed, but often with a point about an inch farther down. The presence of egophony is in no way distinctive of pleuritic effusions and may be heard occasionally over solidified lung.

XIV. PHENOMENA PECULIAR TO PNEUMOHYDROTHORAX AND PNEUMOPYOTHORAX

1. *Succussion Sounds*

Now and then a patient consults a physician, complaining that he hears noises inside him as if water were being shaken about. One such patient expressed himself to me to the effect that he felt "like a half-empty bottle." In the chest of such a patient, if one presses the ear against any portion of the thorax and then shakes the whole patient strongly (succussion), one may hear loud splashing sounds due to air and fluid within. The sound itself is often miscalled "succussion." Such sounds are absolutely diagnostic of the presence of *both air and fluid.* Very frequently they may be detected by the physician when the patient is not aware of their presence. Occasionally the splashing of the fluid within may be felt as well as heard. It is essential, of course, to distinguish splashing due to the presence of air and fluid in the pleural cavity from similar sounds produced in the stomach, but this is not at all difficult in the majority of cases. It is a bare possibility that succussion sounds may be due to the presence of air and fluid in the pericardial cavity, or in the stomach or gut escaped into the thorax through a relaxed or ruptured diaphragm. In accident cases this possibility must be remembered.

It is important to remember that splashing is never to be heard in simple pleuritic effusion or hydrothorax. The presence of air, as well as liquid, in the pleural cavity is absolutely essential to the production of succession sounds.[1]

2. *Metallic Tinkle or Falling-Drop Sound*

When listening over a pleural cavity which contains both air and fluid, one occasionally hears a liquid, tinkling sound, due possibly to the impact of a drop of liquid falling from the relaxed lung above into the accumulated fluid at the bottom of the pleural cavity, but probably to râles produced in the tissues around the cavity. It is stated that this physical sign may in rare cases be observed in large sized phthisical cavities as well as in pneumohydrothorax and pneumopyothorax.

3. *The Lung-Fistula Sound*

When a perforation of the lung occurs below the level of the fluid accumulated in the pleural cavity, bubbles of air may be forced out from the lung and up through the fluid with a sound reminding one of that made by children when blowing soap-bubbles. I have never heard this sound.

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[1] It is well for the student to try for himself the following experiment, which I have found useful in impressing these facts upon the attention of classes in physical diagnosis: Fill an ordinary rubber hot-water bag to the brim with water. Invert it and squeeze out forcibly a certain amount (perhaps half) of the contents, by grasping the upper end of the bag and compressing it. While the water is thus being forced out, screw in the nozzle of the bag. Now shake the whole bag, and it will be found impossible to produce any splashing sounds owing to the fact that there is no air in the bag. Unscrew the nozzle, admit air, and then screw it in again. Now shake the bag again and loud splashing will be easily heard.

CHAPTER IX

AUSCULTATION OF THE HEART

I. "VALVE AREAS"

In the routine examination of the heart, most observers listen in four places:

(1) At the apex of the heart in the fifth intercostal space near the nipple, the "*mitral area.*"

(2) In the second left intercostal space near the sternum, the "*pulmonic area.*"

(3) In the second right intercostal space near the sternum, the "*aortic area.*"

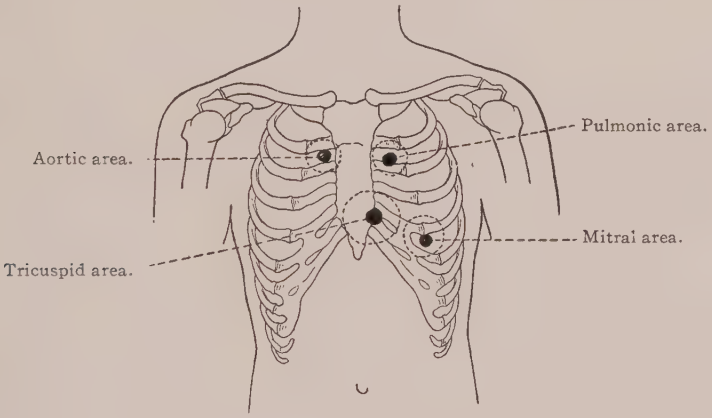

FIG. 125.—The Valve Areas.

(4) At the bottom of the sternum near the ensiform cartilage, the "*tricuspid area.*"

These points are represented in Fig. 125 and are known as "*valve areas.*" They do *not* correspond to the anatomical position of any one of the four valves, but experience has shown that most of the sounds heard best at the apex can be proved (by post-mortem examination) to be produced at the mitral orifice. They are probably transmitted through the papillary muscle whose base or insertion is near the apex region. Similarly, sounds heard best in the second left intercostal space are proved to be produced at the pulmonary orifice;

those which are loudest at the second right intercostal space to be produced at the aortic orifice;[1] more often, however, aortic murmurs are best heard over the third and fourth left costal cartilages ("secondary aortic area"). I can recognize no tricuspid murmurs.

II. The Normal Heart Sounds

A glance at Fig. 126, which represents the anatomical positions of the four valves above referred to, illustrates what I said above; namely, that the traditional valve areas do not correspond at all with the anatomical position of the valves. If now we listen in the "*mitral area,*" that is, in the region of the apex impulse of the heart, keeping at the same time one finger on some point at which the cardiac impulse is palpable, one hears with each outward thrust of the heart a low, dull sound, and in the period between the heart beats a second sound, shorter and sharper in quality.[2]

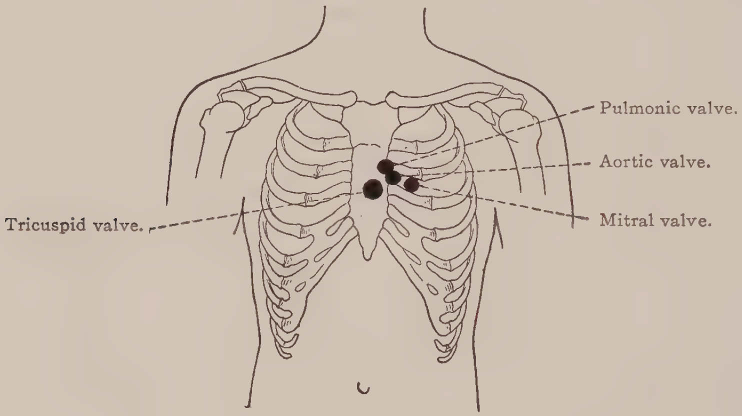

Fig. 126.—Anatomical Position of the Cardiac Valves.

That which occurs with the cardiac impulse is known as the *first sound;* that which occurs between each two beats of the heart is known as the *second sound.* The second sound is generally believed to be due to the closure of the semilunar valves. The cause of the first sound has been a most fruitful source of discussion, and no one explanation of it can be said to be generally received. Perhaps the most commonly accepted view attributes the first or systolic sound of the heart to a combination of two elements—

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[1] For the exceptions to this rule, see below, page 224.

[2] The first sound of the heart, as heard at the apex, may be imitated by holding a linen handkerchief by the corners and suddenly tautening one of the borders. To imitate the second sound, use one-half the length of the border instead of the whole.

(*a*) The contraction of the heart muscle itself.

(*b*) The sudden tautening of the mitral curtains.

Following the second sound there is a pause corresponding to the diastole of the heart. Normally this pause occupies a little more time than the first and second sounds of the heart taken together. In disease it may be much shortened.

The first sound of the heart is not only longer and duller than the second (it is often spoken of as "booming" in contrast with the "snapping" quality of the second sound), but is also considerably more intense, so that it gives us the impression of being accented like the first syllable of a trochaic rhythm. After a little practice one grows so accustomed to this rhythm that one is apt to rely upon his appreciation of the rhythm alone to identify the *first* or systolic sound. *This is, however, an unsafe practice and leads to many errors.* Our impression as to which of the sounds of each cardiac cycle corresponds to systole should always be verified either by sight or touch. We must either see or feel the cardiac impulse and assure ourselves that it is synchronous with the heart sound which we take to be systolic.[1] This point is of especial importance in the recognition and identification of cardiac murmurs, as will be seen presently.

So far, I have been describing the normal heart sounds heard in the "mitral area," that is, at the apex of the heart. If now we listen over the pulmonary area (in the second left intercostal space), we often find that the rhythm of the sounds has changed and that here the stress seems to fall upon the "second sound," *i.e.*, that corresponding to the beginning of diastole; in other words, the first sound of the heart is here heard more feebly and the second sound more distinctly. The sharp, snapping quality of the latter is here even more marked than at the apex, and despite the feebleness of the first sound in this area we can usually recognize its relatively dull and prolonged quality.

Over the traditional *aortic area* (*i.e.*, the second right space) the rhythm is the same as in the pulmonary area, although the second sound may be either stronger or weaker than the corresponding sound on the other side of the sternum (see below, p. 176).

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[1] When the cardiac impulse can be neither seen nor felt, the pulsation of the carotid will generally guide us. The radial pulse is not a safe guide.

Over the tricuspid area one hears sounds practically indistinguishable in quality and in rhythm from those heard at the apex.[1]

When the chest walls are thick and the cardiac sounds feeble, it may be difficult to hear them at all. In such cases the heart sounds may be heard much more distinctly if the patient leans forward and toward his own left. Such a position of the body also renders it easier to map out the outlines of the cardiac dulness by percussion if we allow for the swing of the heart to the left.

In cardiac neuroses, thyrotoxicoses and during excitement or emotional strain, the first sound at the apex is not only very loud but has often a curious *metallic reverberation* ("cliquetis metallique") corresponding to the trembling, jarring cardiac impulse (often mistaken for a thrill) which palpation reveals.

III. Modifications in the Intensity of the Heart Sounds

It has already been mentioned that in young persons with thin, elastic chests, the heart sounds are heard with greater intensity than in older persons whose chest walls are thicker and stiffer. In obese, indolent adults it is sometimes difficult to hear any heart sounds at all, while in young persons of excitable temperament the sounds may have a very intense and ringing quality. Under diseased conditions either of the heart sounds may be increased or diminished in intensity. I shall consider

1. *The First Sound at the Apex (sometimes Called the Mitral First Sound)*

(*a*) Increase in the intensity of the first sound at the apex of the heart occurs in any condition which causes the heart to act with unusual degree of force, such as thyrotoxicosis, exertion, or excitement. In the earlier stages of infectious fevers a similar increase in the intensity of this sound may sometimes be noted. Hypertrophy of the left ventricle sometimes has a similar effect upon the sound, but less often than one would suppose, while weakening of the left ventricle, contrary to what one would suppose, is not infrequently associated with a loud, forcible first sound at the apex. In mitral stenosis the first sound is usually very intense, and is often spoken of as a "thumping first sound" or as a "sharp slap."

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[1] A third heart sound (or reduplication of the second sound) is audible on careful auscultation in a considerable proportion of healthy young individuals—especially if they lie on the left side. Barié described it in 1893 (*Semaine méd.*, 1893, xiii, 474), and Thayer (*Boston Med. & Surg. Journ.*, May 7, 1908) has recently recalled it to notice, believing it due to "the sudden tension of the mitral and perhaps at times tricuspid valves occurring at the end of the first and most rapid phase of diastole." This is probably identical with the double second sound of mitral stenosis and with one of the types of gallop rhythm. No diagnostic significance is as yet clearly associated with it.

(*b*) Shortening and weakening of the first sound at the apex.

In the course of continued fevers and especially in typhoid fever the granular degeneration which takes place in the heart muscle is manifested by a shortening and weakening of the first sound at the apex, so that the two heart sounds come to seem much more alike than usual. In the later stages of typhoid, the first sound may become almost inaudible. The sharp "valvular" quality, which one notices in the first apex sound under these conditions, has been attributed to the fact that weakening of the myocardium has caused a suppression of one of the two elements which go to make up the first sound, namely, the muscular element, so that we hear only the short, sharp sound due to the tautening of the mitral curtains. Arterio-sclerosis, or any other change which tends to enfeeble the heart wall, produces a weakening and shortening of the first sound similar to that just described. Simple weakness in the mitral first sound without any change in its duration or pitch may be due to fatty overgrowth of the heart, to emphysema or pericardial effusion in case the heart is covered by the distended lung or by the accumulated fluid. All in all, however, these changes in the first sound are of very little diagnostic use.

(*c*) Doubling of the first sound at the apex.

It is not uncommon in healthy hearts to hear in the region of the apex impulse a doubling of the first sound so that it may be suggested by pronouncing the syllables "turrupp" or "trupp." In health this is especially apt to occur at the end of expiration. In disease it is associated with many different conditions involving an increase in the work of one or the other side of the heart. It seems to be unusually frequent in the weakened heart of nephritis and arteriosclerosis, but is not of much importance.

2. *Modifications in the Second Sounds as Heard at the Base of the Heart*

(*a*) *Physiological Variations*

The relative intensity of the pulmonic second sound, when compared with the second sound heard in the conventional aortic area, varies a great deal at different periods of life. Attention was first called to this by Vierordt,[1] and it has of late years been recognized by the best authorities on diseases of the heart.

The work of Dr. Sarah R. Creighton, done in my clinic during the summer of 1899, showed that in 90 per cent. of healthy children under ten years of age, the pulmonic second sound is louder than the aortic. In the next decade (from the tenth to the twentieth year) the pulmonic second sound is louder in two-thirds of the cases. About half of 207 cases, between the ages of twenty and twenty-nine, showed an accentuation of the pulmonic second, while after the thirtieth year the number of cases showing such accentuation became smaller with each decade, until after the sixtieth year we found an accentuation of the *aortic second in sixty-six out of sixty-eight cases* examined. These facts are exhibited in tabular form in Figs. 126a and 127, and appear to show that the relative intensity of the two sounds in the aortic and pulmonic arteries depends primarily upon the age of the individual, the pulmonic

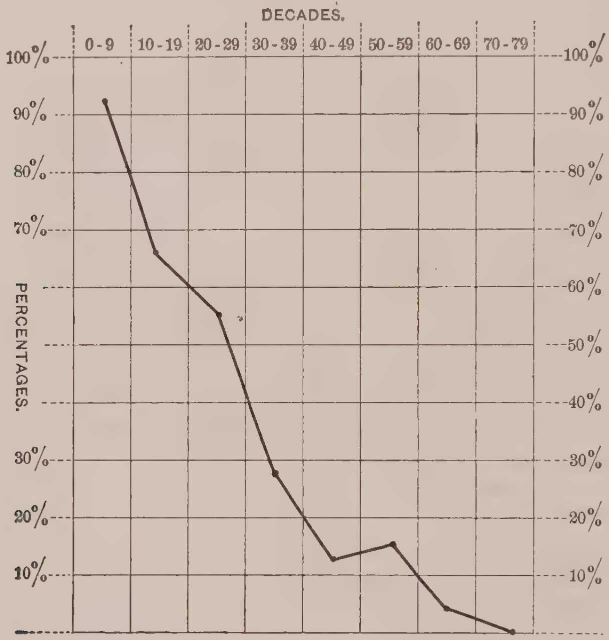

FIG. 126a.—Showing the Per Cent. of Accentuated Pulmonic Second Sound in Each Decade. Based on 1,000 cases.

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[1] Vierordt: "Die Messung der Intensität der Herztöne" (Tübingen, 1885). See also Hochsinger, "Die Auscultation des kindlichen Herzens;" Gibson, "Diseases of the Heart" (1898); Rosenbach, "Diseases of the Heart" (1900); Allbutt, "System of Medicine."

sound predominating in youth and the aortic in old age, while in the period of middle life there is relatively little discrepancy between the two. It is, therefore, far from true to suppose that we can obtain evidence of a pathological increase in the intensity of either of the second sounds at the base of the heart simply by comparing it with the other. Pathological accentuation of the pulmonic second sound must mean a greater loudness of this sound *than should be expected at the age of the patient in question*, and not simply a greater intensity than that of the aortic second sound. The same observation obviously applies to accentuation of the aortic second sound.

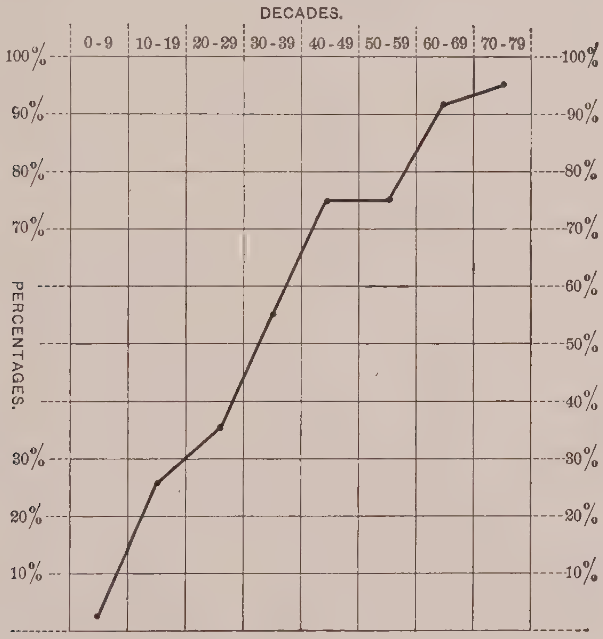

FIG. 127.—Showing the Per Cent. of Accentuated Aortic Second Sound in Each Decade. Based on 1,000 cases.

Both the aortic and the pulmonic second sounds are sometimes very intense during emotional excitement, in Graves' disease, after muscular exertion, and sometimes without any obvious cause.

(*b*) *Pathological Variations*

Accentuation of the Pulmonic Second Sound

Pathological accentuation of the second sound occurs especially in conditions involving a backing up of blood in the lungs, such as occurs in stenosis and insufficiency of the mitral valve, or in obstructive disease of the lungs (bronchitis, phthisis, chronic interstitial pneumonia). Indirectly, accentuation of the pulmonic second sound points to hypertrophy of the right ventricle, since without such hypertrophy the work of driving the blood through the obstructed lung could not long be performed. If the right ventricle becomes weakened the accentuation of the pulmonic second sound is no longer heard.

Weakening or Absence of the Pulmonic Second Sound

Weakening of the pulmonic second sound is a very serious symptom, sometimes to be observed in cases of pneumonia or cardiac disease near death.

Pulmonary stenosis also weakens or abolishes the second sound, and in many other types of congenital heart disease the pulmonic, as well as the aortic, second sound in inaudible. I have found it absent in aortic stenosis. Indeed, I think it may be stated that *with any very loud systolic murmur at the base of the heart, we may find the pulmonic second sound gone; why I do not know.*

Accentuation of the Aortic Second Sound

I have already shown that the aortic second sound is louder than the corresponding sound in the pulmonary area in almost every individual over sixty years of age and in most of those over forty. A still greater intensity of the aortic second sound occurs—

(*a*) In nephritis, arterio-sclerosis, or any condition which increases arterial tension and so throws an increased amount of work upon the left ventricle. Directly, therefore, a pathologically loud aortic sound points to increased resistance in the peripheral arteries and indirectly to hypertrophy of the left ventricle.

(*b*) A similar increase in the aortic second sound occurs in aneurism or sclerosis of the arch even without high blood-pressure.

Diminution in the Intensity of the Aortic Second Sound

Whenever the amount of blood thrown into the aorta by the contraction of the left ventricle is diminished, as is the case especially in mitral stenosis and in most cases of aortic stenosis, the aortic second sound is weakened so that at the apex it may be inaudible. A similar effect is produced by any disease which weakens the walls of the left ventricle. Relaxation of the peripheral arteries has the same effect. In conditions of collapse the aortic second sound may be almost or quite inaudible.

In persons past middle life the second sounds are often louder in the third or fourth interspace than in the second, so that if we listen only in the second space we may gain the false impression that the second sounds are feeble.

Accentuation of both the second sounds at the base of the heart may occur in health from nervous causes or when the lungs are retracted by disease so as to uncover the conus arteriosus and the aortic arch. Under these conditions the second sound may be seen and felt as well as heard. In a similar way, an apparent increase in the intensity of either one of the second sounds at the base of the heart may be produced by a retraction of one or the other lung.

Summary.—(1) The *mitral first sound* is increased by whatever induces increased vigor of the heart, and among valvular diseases, especially by mitral stenosis. It is weakened or reduplicated when the heart is weakened. Any of these changes may occur temporarily from physiological causes.

(2) The *pulmonic second sound* is usually more intense than the aortic in children and up to early adult life. Later the aortic second sound predominates. Pathological accentuation of the second pulmonic sound usually points to obstruction in the pulmonary circulation (mitral disease, etc.). Weakening of the pulmonic second means failure of the right ventricle and is serious.

(3) The *aortic second sound* is increased pathologically by any cause which increases the work of the left ventricle (arterio-sclerosis, chronic nephritis). It is diminished when the blood stream, thrown into the aorta by the left ventricle, is abnormally small (mitral disease, cardiac failure).

(4) Changes in the tricuspid sounds are rarely recognizable, while changes in the first sound as heard in aortic and pulmonic areas have little practical significance.

3. *Modifications in the Rhythm of the Cardiac Sounds*

(1) Whenever the walls of the heart are greatly weakened by disease—for example, in the later weeks of a case of typhoid fever—the diastolic pause of the heart is shortened so that the cardiac sounds follow each other almost as regularly as the ticking of a clock; hence the term "*tick-tack* heart." As this rhythm is not unlike that heard in the fœtal, the name of "*embryocardia*" is sometimes applied to

it. The "tick-tack" rhythm may be heard in paroxysmal tachycardia, in thyrotoxicosis, infectious diseases and in any condition leading to collapse.

(2) The term *gallop rhythm* refers to conditions in which three sounds are audible at or near the apex.

(*a*) The commoner type is the *presystolic gallop rhythm* in which an extra sound occurs just before the ordinary first sound of the heart (practically a double first sound with accent on the second half). Such a rhythm may occur temporarily in any heart which is excited or overworked from any cause, but when permanent it is usually a sign of grave cardiac weakness (nephritic cases, arterio-sclerosis, chronic valvular disease, goitre, etc.).

(*b*) *Protodiastolic Gallop Rhythm* (doubling of the second sounds at the base of the heart). The extra sound is not easily distinguished from the so-called "third heart sound" referred to on page 217 and *most constantly heard in the early stages of mitral stenosis*, but the interval between the two parts is longer in the latter case.

At the end of a long inspiration this change may be observed in almost any healthy persons if one listens at the base of the heart. It is still better brought out after muscular exertion or by holding the breath.

In mitral stenosis the double diastolic sound is also to be heard at the apex, and in the diagnosis of this disease this "double shock sound" during diastole may be an important piece of evidence, and may sometimes be felt and seen as well as heard. Just what its mechanism is, is disputed. Except in mitral stenosis, it has no especial clinical significance.

4. *Metallic Heart Sounds*

The presence of air in the immediate vicinity of the heart, as, for example, in pneumothorax or in gaseous distention of the stomach or intestine, may impart to the heart sounds a curious metallic quality such as is not heard under any other conditions.

5. *"Muffling," "Prolongation," or "Unclearness" of the Heart Sounds*

These terms are not infrequently met with in literature, but their use should, I think, be discontinued. The facts to which they refer should be explained either as faintness of the heart sounds, due to the causes above assigned, or as faint, short murmurs. In their present usage such terms as "muffled" or "unclear" heart sounds represent chiefly an unclearness in the mind of the observer as to just what it is that he hears, and not any one recognized pathological condition in the heart.

IV. Sounds Audible over the Peripheral Vessels

(1) The normal heart sounds are in adults audible over the carotids and over the subclavian arteries. In childhood and youth only the second heart sound is thus audible.

(2) In about 7 per cent. of normal persons a systolic sound can be heard over the femoral artery. This sound is obviously not transmitted from the heart, and is usually explained as a result of the sudden systolic tautening of the arterial wall.

In aortic regurgitation this arterial sound is almost always audible not only in the femoral but in the brachial and even in the radial, and its intensity over the femoral becomes so great that the term "pistol-shot" sound has been applied to it. In fevers, exophthalmic goitre, and other diseases involving a large pulse pressure, a similar arterial sound is to be heard much more frequently than in health.

CHAPTER X

AUSCULTATION OF THE HEART: CONTINUED

CARDIAC MURMURS

I. Terminology

The word "*murmur*" is one of the most unfortunate of all the terms used in the description of physical signs. No one of the various blowing, whistling, rolling, rumbling, or piping noises to which the term refers, sounds anything like a "murmur" in the ordinary sense of the word. Nevertheless, it does not seem best to try to replace it by any other term. The French word "*souffle*" is much more accurate and has become to some extent Anglicized. Under the head of cardiac murmurs are included all abnormal sounds produced within the heart itself. Pericardial friction sounds and those produced in that portion of the lung or pleura which overlies the heart are not considered "murmurs."

II. Mode of Production

With rare exceptions all cardiac murmurs are produced at or near one of the valve orifices, either by disease of the valves themselves resulting in shrivelling, thickening, stiffening, and narrowing of the valve curtains, by a stretching of the orifice into which the valves are inserted, or by disease of the aortic arch.

Diseases of the valves themselves may lead to the production of murmurs:

(*a*) When the valves are too stiff and short to close (incompetence, insufficiency, or *regurgitation*).

(*b*) When the valves fail to open at the proper time (*stenosis* or obstruction).

(*c*) When the surfaces of the valves or of the parts immediately adjacent are *roughened* so as to prevent the smooth flow of the blood over them.

(*d*) When the orifice which the valves are meant to close is dilated as a result of *dilatation* of the heart chamber of which it forms the entrance or exit. The valves themselves cannot enlarge to keep pace with the enlargement of the orifice, and hence no longer suffice to reach across it.

The presence of any one of these lesions gives rise to eddies in the blood current and thereby to the abnormal sounds to which we give the name murmurs.[1] (See Figs. 129, 130 and 131.) When valves fail to close and so allow the blood to pass back through them, we speak of the lesion as *regurgitation, insufficiency, or incompetence;* if, for example, the aortic valves fail to close after the left ventricle has thrown a column of blood into the aorta, some of this blood regurgitates through these valves into the ventricle from which it has just been expelled, and we speak of the lesion as "*aortic regurgitation,*" and of the murmur so produced as an *aortic regurgitant murmur* or a murmur of aortic regurgitation. If the mitral valve fails to open properly to admit the blood which should flow during diastole from the left auricle into the left ventricle, we speak of the condition as *mitral stenosis* or *mitral obstruction.* A similar narrowing of the aortic

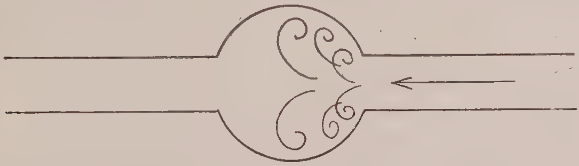

FIG. 128.—Diagram to Illustrate the Production of a Cardiac Murmur Through Regurgitation from the Aorta or in an Aneurismal Sac. The arrow shows the direction of the blood current and the curled lines the audible blood eddies.

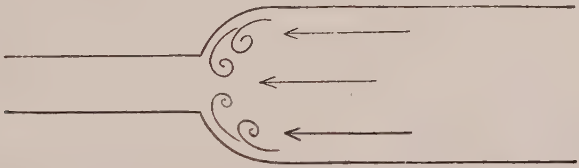

FIG. 129.—Diagram to Illustrate the Production of a Cardiac Murmur Through Stenosis of a Valve-Orifice.

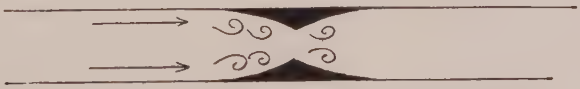

FIG. 130.—Diagram to Illustrate the Production of Cardiac Murmurs Through Roughening of a Valve.

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[1] The method by which functional murmurs are produced will be discussed later. (See page 191.)

valves such as to hinder the egress of blood during the systole of the left ventricle is known as *aortic stenosis* or obstruction. Valvular lesions of the right side of the heart (tricuspid and pulmonic valves) are comparatively rare, but are produced and named in a way similar to those just described.

The facts most important to know about a murmur are:

(1) Its place in the cardiac cycle.
(2) Its point of maximum intensity.
(3) The area over which it can be heard.
(4) The effects of exertion, respiration, or position upon it.
Less important than the above are:
(5) Its intensity.
(6) Its quality.
(7) Its length.
(8) Its relation to the normal sounds of the heart.

Each of these points will now be taken up in detail:

(1) *Time of Murmurs.*—The first and most important thing to ascertain regarding a murmur is its relation to the normal cardiac cycle; that is, whether it occurs during systole or during diastole, or in case it does not fill the whole of one of those periods, in what part of systole or diastole it occurs. It must be borne in mind that the period of systole is considered as lasting from the beginning of the first sound of the heart up to the occurrence of the second sound, while diastole lasts from the beginning of the second sound until the beginning of the first sound in the next cycle. Any murmur occurring with the first sound of the heart, or at the time whan the first sound should take place, or in any part of the period intervening between the first sound and the second, is held to be *systolic*. Murmurs which distinctly follow the first sound or do not begin until the first sound is ended are known as *late systolic* murmurs.

On the other hand, it seems best, for reasons to be discussed more in detail later on, not to give the name of *diastolic* to all murmurs which occur within the diastolic period as above defined. Murmurs which occur during the last part of diastole and which run up to the first sound of the next cycle are usually known as "*presystolic*" murmurs. All other murmurs occurring during diastole are known as *diastolic*.

The commonest of all the errors in the diagnosis of disease of the heart is to mistake systole for diastole, and thereby to misinterpret the significance of a murmur heard during those periods. This mistake would never happen if we were always careful to make sure, by means of sight or touch, just when the systole of the heart occurs. This may be done by keeping one finger upon the apex impulse of the heart or upon the carotid artery while listening for murmurs, or, in case the apex impulse or the pulsations of the carotid are better seen than felt, we can control by the eye the impressions gained by listening. It is never safe to trust our appreciation of the cardiac rhythm to tell us which is the first heart sound and which the second. The proof of this statement is given by the numberless mistakes made through disregarding it. Equally untrustworthy as a guide to the time of systole and diastole is the radial pulse, which follows the cardiac systole at an interval just long enough to mar our calculations.

(2) *Localizations of Murmurs.*—To localize a murmur is to find its point of maximum intensity, and this is of the greatest importance in diagnosis. Long experience has shown that murmurs heard loudest in the region of the apex beat (whether this is in the normal situation or displaced), are in the majority of cases produced at the mitral valve. In about five per cent. of the cases mitral murmurs may be best heard at a point midway between the position of the normal cardiac impulse and the ensiform cartilage, or, (rarely), an inch or two above this situation.

Murmurs heard most loudly in the second left intercostal space are usually produced at the pulmonic orifice or just above it in the conus arteriosus. Many congenital heart defects produce murmurs here.

Murmurs produced at the aortic orifice may be heard best in the aortic area, but in a large proportion of cases are loudest on the other side of the sternum at or about the situation of the fourth left costal cartilage. Sometimes they are best heard at the apex of the heart in the axilla or over the lower part of the sternum (see below, Fig. 149).

(3) *Transmission of Murmurs.*—If a murmur is audible over several valve areas, the questions naturally arise: "How are we to know whether we are dealing with a single valve lesion or with several? Is this one murmur or two or three murmurs?" Obviously the question can be asked only in case the murmur which we find audible in various places occupies everywhere the same time in the cardiac cycle. It must, for example, be everywhere systolic or everywhere diastolic. A systolic murmur at the apex cannot be supposed to point to the same lesion as a diastolic murmur, no matter where the latter is heard. But, if we hear a systolic murmur in various parts of the chest, say over the aortic, mitral, and pulmonic regions, how are we to know whether the sound is simple or compound, whether produced at one valve orifice or at several?

This question is sometimes difficult to answer, and in a given case skilled observers may differ in their verdict, but, as a rule, the difficulty may be overcome as follows:

(1) Experience and post-mortem examination have shown that the murmur produced by each of the valvular lesions has its own characteristic area of propagation, over which it is heard with an intensity which regularly diminishes as we recede from a *maximum* whose seat corresponds with some one of the valve areas just described. These areas of propagation are shown in Figs. 142, 147, and 150. Any murmur whose distribution does not extend beyond one of these areas, and which steadily and progressively diminishes in intensity as we move away from the valve area over which it is loudest, may be assumed to be due to a single valve lesion and no more. Provided but one valve is diseased, this course of procedure gives satisfactory results.

(2) When several valves are diseased and several murmurs may be expected, it is best to start at some one valve area, say in the mitral or apex region, and move the stethoscope one-half an inch at a time toward one of the other valve areas, noting the intensity of any murmur we may hear at each of the different points passed over. As we move toward the aortic area, we may get an impression best expressed by Fig. 133. As we go on in the same direction the murmur may first grow fainter then louder (and perhaps change in pitch and quality as well) until a maximum is reached at the aortic area, beyond which the murmur again fades out.

These facts justify us in *suspecting* that we are dealing with two murmurs, one produced at the tricuspid and one at the mitral orifice. The suspicion is more likely to be correct if there has been a change in the pitch and quality of the murmur as we neared the second orifice, and may be confirmed by the discovery of other evidences of a double lesion. But *no diagnosis is satisfactory which rests on the evidence of murmurs alone.* Changes in the size of the heart's chambers or in the pulmonary or peripheral circulations are the most important facts in the case. Nevertheless the effort to ascertain, and graphically to represent the intensity of cardiac murmurs as one listens along the line connecting the valve areas has its value. An "hour-glass" murmur, such as that represented in Fig. 133 generally means *two-valve* lesions. An "hour-glass" shape may be found to represent the auditory facts as we move from the mitral to the pulmonic or to the aortic areas (see Fig. 131) and, as in the previous case, arouses our suspicion that more than one valve is diseased.

It must not be forgotten, however, that "a murmur may travel some distance underground and emerge with a change of quality" (Allbutt). This is especially true of aortic systolic murmurs, which are often heard well at the apex and at the aortic area, and faintly in the intervening space, probably owing to the interposition of the right ventricle.

In such cases we fall back upon the other relevant facts shown by *x*-ray, blood-pressure, inspection, palpation, and percussion, upon the condition of the lungs and arteries as shown in the other symptoms and signs of the case (dropsy, cough, etc.), and upon the etiology.

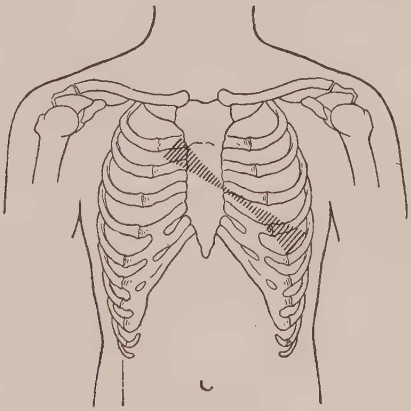

FIG. 131.—Mitral Disease and Aortic Stenosis. The systolic murmur is loudest at the extremities of the shaded area; and faintest at its "waist."

(3) *Intensity of Murmurs.*—Sometimes murmurs are so loud that they are audible to the patient himself or even at some distance from the chest. In one case I was able to hear a murmur eight feet from the patient. Such cases are rare and usually not serious, for the gravity of the lesion is not at all proportional to the loudness of the murmur; indeed, other things being equal, loud murmurs are less serious than faint ones, provided we are sure we are dealing with organic lesions. (On the distinction between the organic and functional murmurs, see below, p. 190.)

A loud murmur means a powerful heart driving the blood strongly over the diseased orifice. When the heart begins to fail, the intensity of the murmur proportionately decreases because the blood does not flow swiftly enough over the diseased valve to produce as loud a sound as formerly. The gradual disappearance of a murmur known to be due to a valvular lesion is, therefore a very grave sign, and its reappearance revives hope. Patients are not infrequently admitted to a hospital with valvular heart trouble which has gone on so long that the muscle of the heart is no longer strong enough to produce a murmur as it pumps the blood over the diseased valve. In such a case, under the influence of rest and cardiac tonics, one may observe the development of a murmur as the heart wall regains its power, and the louder the murmur becomes the better the condition of the patient. On the other hand, when the existence of a valvular lesion has been definitely determined, and yet the compensation remains perfectly good (for example, in the endocarditis occurring in children in connection with chorea), an increase in the loudness of the murmur may run parallel with the advance in the valvular lesion.

In general the most important point about the intensity of a murmur is its *increase or decrease while under observation*, and not its loudness at any one time.

(4) *Quality of Heart Murmurs.*—It has been already mentioned that the quality of a heart murmur is never anything like the sound which we ordinarily designate by the word "murmur." The commonest type of heart murmur has a blowing quality whence the old name of "*bellows sound.*" The sound of the letter "f" prolonged is not unlike the quality of certain murmurs. *Blowing murmurs* may be low-pitched like the sound of air passing through a large tube, or high-pitched approaching the sound of a whistle. This last type merges into that known as the *musical* murmur, in which there is a definite musical sound whose pitch can be identified. *Rasping* or tearing sounds often characterize the louder varieties of murmurs.

Finally, there is one type of sound which, though included under the general name murmur, differs entirely from any of the other sounds just described. This is the "*presystolic roll,*" which has a *rumbling* or *blubbering* quality or may remind one of a short drum-roll. This murmur is always presystolic in time and usually associated with obstruction at the mitral or tricuspid valves. Not infrequently some part of a cardiac murmur will have a musical quality while the rest is simply blowing or rasping in character. Musical murmurs do not give us evidence either of an especially serious or especially mild type of disease. Their chief importance consists in the fact that they rarely exist without some valve lesion,[1] and are, therefore, of use in excluding the type of murmur known as "*functional,*" presently to be discussed, and not due to valve disease. Very often rasping murmurs are associated either with the calcareous deposit upon a valve or very marked narrowing of the valve orifice.

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[1] Rosenbach holds that they may be produced by adhesive pericarditis.

Murmurs may be accented at the beginning or the end; that is, they may be of the crescendo type, growing louder toward the end, or of the decrescendo type with their maximum intensity at the beginning.

(5) *Length of Murmurs.*—Murmurs may occupy the whole of systole, the whole of diastole, or only a portion of one of these periods, but no conclusions can be drawn as to the severity of the valve lesion from the length of the murmur. A short murmur, especially if diastolic, may be of very serious prognostic import.

(6) *Relations to the Normal Sounds of the Heart.*—Cardiac murmurs may or may not replace the normal heart sounds. They may occur simultaneously with one or both sounds or between the sounds. These facts have a certain amount of significance in prognosis. Murmurs which entirely replace cardiac sounds usually mean a severer disease of the affected valve than murmurs which accompany, but do not replace, the normal heart sounds. Late systolic murmurs, which occur between the first and the second sound, are usually associated with a relatively slight degree of valvular disease. Late diastolic murmurs, on the other hand, have no such favorable significance.

(7) *Effects of Position, Exercise, and Respiration upon Cardiac Murmurs.*—Almost all cardiac murmurs are affected to a greater or less extent by the position which the patient assumes while he is examined. Systolic murmurs which are inaudible while the patient is in a sitting or standing position may be quite easily heard when the patient lies down. On the other hand, a presystolic roll which is easily heard when the patient is sitting up may nearly disappear when he lies down. Diastolic murmurs are relatively little affected by the position of the patient, but in the majority of cases are somewhat louder in the upright position.

The effects of exercise may perhaps be fitly mentioned here. Feeble murmurs may altogether disappear when the patient is at rest, and under such circumstances may be made easily audible by getting the patient to walk briskly up and down the room a few times. Such lesions are usually comparatively slight.[1] On the other hand, murmurs which become more marked as a result of rest are generally of the severest type.

Organic murmurs are usually better heard at the end of expiration and become fainter during inspiration as the expanding lung covers the heart. This is especially true of those produced at the mitral valve, and is in marked contrast with the variations of functional murmurs which are heard chiefly or exclusively at the end of inspiration.

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[1] For exception to this see below, page 208.

(8) *Sudden Metamorphosis of Murmurs.*—In acute endocarditis, when vegetations are rapidly forming and changing their shape upon the valves, murmurs may appear and disappear very suddenly. This metamorphosing character of cardiac murmurs, when taken in connection with other physical signs, may be a very important factor in the diagnosis of acute endocarditis. In a similar way relaxation or rupture of one of the tendinous cords, occurring in the course of acute endocarditis, may effect a very sudden change in the auscultatory phenomena.

III. Functional Murmurs

Not every murmur which is to be heard over the heart points to disease either in the valves or in the orifices of the heart. Perhaps the majority of all murmurs are thus unassociated with valvular disease, and to such the name of "accidental," "functional," or "hæmic" murmurs has been given. The origin of these "functional" murmurs has given rise to an immense amount of controversy, and it cannot be said that any one explanation is now generally agreed upon. To me, the most plausible view is that which regards most of them as due either to a temporary or permanent dilatation of the conus arteriosus, or to pressure or suction exerted upon the overlapping lung margins by the cardiac contractions. This explains only the systolic functional murmurs, which make up ninety-nine per cent. of all functional murmurs. The diastolic functional murmurs, which undoubtedly occur, although with exceeding rarity, are probably due to stretching of the aortic ring or to sounds produced in the veins of the neck and transmitted to the vena cava.

Characteristics of Functional Murmurs.—(1) Almost all functional murmurs are systolic, as has before been mentioned.

(2) The majority of them are heard best over the pulmonic valve in the second left intercostal space. From this point they are transmitted in all directions, and are frequently to be heard, although with less intensity, in the aortic and mitral areas. Not infrequently they may have their maximum intensity in one of the latter positions.

(3) As a rule, they are very soft, *short*, and blowing in quality, though exceptionally they may be loud and rough. They almost never extend through the whole of systole.

(4) They are usually not associated with evidence of enlargement of the heart nor with accentuation of the pulmonic second sound.[1]

(5) They are usually louder at the end of inspiration.

(6) They are usually heard over a very limited area, but to this rule there are exceptions.

(7) They are especially evanescent in character; for example, they may appear at the end of a hard run or boat race or during an attack of fever, and disappear within a few days or hours. Respiration, position, and exercise produce greater variations in them than in "organic" murmurs.

(8) They are especially apt to be associated with *anæmia*, although the connection between anæmia and functional heart murmurs is by no means as close as has often been supposed. The severest types of anæmia, for example pernicious anæmia, may not be accompanied by any murmur, while, on the other hand, typical functional murmurs are often heard in patients whose blood is normal, and even in full health. Yet in three cases of intense anæmia I have heard *diastolic* murmurs loudest at the fourth left costal cartilage and leading to a diagnosis of aortic regurgitation. At autopsy the aortic valves were in each case sound, and I am at a loss to account for the murmurs.[2]

The distinctions between organic and functional heart murmurs may be summed up as follows:

Organic murmurs may occupy any part of the cardiac cycle; if systolic, they are often transmitted either into the axilla and back, or into the great vessels of the neck; they are usually associated with evidences of cardiac enlargement and changes in the second sounds at the base of the heart, as well as with signs and symptoms of stasis in other organs. Organic murmurs not infrequently have a musical or rasping quality, although this is by no means always the case. They are rarely loudest in the pulmonic area and are relatively uninfluenced by respiration, position, or exercise.

Functional murmurs are almost always systolic in time and usually heard with maximum intensity in the pulmonic area. They are rarely transmitted beyond the precordial region and are usually loudest at the end of inspiration. They are not accompanied by evidences of cardiac enlargement or pathological accentuation of the second sounds at the base of the heart, nor by signs of venous stasis or dropsy.

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[1] In chlorosis the second pulmonic sound is often very loud (owing to the retraction of the lungs and uncovering of the conus arteriosus) and associated with a systolic murmur.

[2] Cabot and Locke: *Johns Hopkins Bulletin*, May, 1903.

They are very apt to be associated with anæmia or with some special attack upon the resources of the body (*e.g.*, physical overstrain or fever), and to disappear when such forces are removed. They are usually short and soft in quality; rarely musical. The very rare diastolic functional murmur occurs oftenest, so far as I am aware, in conditions of profound anæmia; *i.e.*, when the hæmoglobin is twenty-five per cent. or less, and of hypertension.

IV. Cardio-Respiratory Murmurs

When a portion of the free margin of the lung is fixed by adhesions in a position overlapping the heart, the cardiac movements may rhythmically displace the air in such piece of lung so as to give rise to sounds which at times closely simulate cardiac murmurs. These conditions are most often to be found in the tongue-like projection of the left lung, which normally overlaps the heart, but it is probably the case that cardio-respiratory murmurs may be produced without any adhesion of the lung to the pericardium under conditions not at present understood. Such murmurs may be heard under the left clavicle or below the angle of the left scapula, as well as near the apex of the heart,—less often in other parts in the chest.

Cardio-respiratory murmurs may be either systolic or diastolic, but in the vast majority cases are systolic. The area over which they are audible is usually a very limited one. They are greatly affected by position and by respiration, and are heard most distinctly, if not exclusively, during inspiration, especially at the end of that act. (This fact is an important aid in distinguishing them from true cardiac murmurs, which are almost always fainter at the end of inspiration.) They are also greatly affected by cough or forced respiration or by holding the breath, whereas cardiac murmurs are relatively little changed thereby. Pressure on the outside of the thorax and in their vicinity may greatly modify their intensity or quality, while organic cardiac murmurs are less influenced by pressure. As a rule, they have the quality of normal *respiratory murmurs, and sound like an inspiration interrupted by each systole of the heart.*[1]

In case the effect of the cardiac movements is exerted upon a piece of lung in which a catarrhal process is going on, we may have systolic or diastolic crackles or squeaks, or any type of respiratory murmur except the bronchial type, since this is produced in solid lung which could not be emptied or filled under the influence of the cardiac movements. Cardio-respiratory murmurs have no special diagnostic significance, and are mentioned here only on account of the importance of not confusing them with true cardiac murmurs. They were formerly thought to indicate phthisis, but such is not the case.

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[1] For the distinction from cog-wheel breathing, see above, page 153.

V. Arterial Murmurs

(1) Roughening or slight dilatation of the arch of the aorta, due to chronic endaortitis, is a frequent cause in elderly people of a systolic murmur, heard best at the base of the heart and transmitted into the vessels of the neck. From cardiac murmurs it is distinguished by the lack of any other evidence of cardiac disease and by the evidence of marked arteriosclerosis in the peripheral vessels (see further discussion under Aortic Stenosis, p. 225, and under Aneurism, p. 261).

(2) A narrowing of the lumen of the subclavian artery, due to some abnormality in its course, may give rise to a systolic murmur heard close below the clavicle at its outer end. The murmur is greatly influenced by movements of the arm and especially by respiratory movements. During inspiration it is much louder, and at the end of a forced expiration it may disappear altogether. Occasionally such murmurs are transmitted through the clavicle so as to be audible above it.

(3) Pressure exerted upon any of the superficial arteries, carotid, femoral, etc.) produces a systolic murmur. Diastolic arterial murmurs are peculiar to aortic regurgitation.

(4) Over the anterior fontanella in infants and over the gravid uterus systolic murmurs are to be heard which are probably arterial in origin.

(5) Thayer has recently described an epigastric murmur in a case of cirrhotic liver.

CHAPTER XI

ESTABLISHMENT AND FAILURE OF COMPENSATION IN VALVULAR DISEASE OF THE HEART

We may discriminate three periods in the progress of a case of valvular heart disease:

(1) The period before the establishment of compensation.
(2) The period of compensation.
(3) The period of failing or ruptured compensation.

I. Compensation not yet Established

In most cases of acute "rheumatic" endocarditis, whether of the relatively benign or of the malignant type, there is a time when the lesion is perfectly recognizable despite the fact that compensatory hypertrophy has not yet occurred. In some cases this period may last for months; the heart is not enlarged, there is no accentuation of either second sound at the base, there is no venous stasis, and our diagnosis must rest solely upon the presence and characteristics of the murmur. For example, in early cases of mitral lesion associated with chorea or rheumatism, the disease may be recognized by the presence of a long or musical murmur heard in the back as well as at the apex and in the axilla. In the earlier stages of aortic regurgitation occurring in young people as a complication of rheumatic fever, there may be absolutely no evidence of the valve lesion except the characteristic diastolic murmur.

II. The Period of Compensation

Valvular disease would, however, soon prove fatal were it not for the occurrence of compensatory hypertrophy of the heart walls. To a certain extent the heart contracts as a single muscle, and increases the size of all its walls in response to the demand for increased work; but as a rule the hypertrophy predominates in one ventricle—that ventricle, namely, upon which especially demand is made for increased power in order to overcome an increased resistance in the vascular circuit which it supplies with blood. Whatever increases the resistance in the lungs brings increased work upon the right ventricle; whatever increases the resistance in the aorta or peripheral arteries increases the amount of work which the left ventricle must do.

Now, any disease of the mitral valve, whether obstruction or leakage, results in engorgement of the lungs with blood, and hence demands an increased amount of work on the part of the right ventricle in order to force the blood through the overcrowded pulmonary vessels; hence it is in mitral disease that we find the greatest compensatory predominance of the right ventricle.

On the other hand, it is obvious that obstruction at the aortic valves or in the peripheral arteries with or without arterio-sclerosis, or nephritis demands an increase in power in the left ventricle, in order that the requisite amount of blood may be forced through arteries of reduced calibre, while if the aortic valve is so diseased that a part of the blood thrown into the aorta by the left ventricle returns into the ventricle, its work is thereby greatly increased, since it has to contract upon a larger volume of blood.

In response to these demands for increased work, the muscular wall of the left ventricle increases in thickness, and compensation is thus established at the cost of an increased amount of work on the part of the heart.[1]

III. Failure of Compensation

Sooner or later in the vast majority of cases, the heart, handicapped as it is by a leakage or obstruction of one or more valves, becomes unable to meet the demands made upon it by the needs of the circulation. Failure of compensation is associated with decrease of muscular tone and thence with weakening of the heart's contraction. Not infrequently recurrent *attacks of "failing compensation" represent a flare-up of a smouldering endocarditis as the accompanying leucocytosis (with or without fever) suggests.* This is especially common in children but occurs also in young adults. Sometimes, however, neither mechanical nor infectious changes can be found. Whatever the cause may be, the result of ruptured compensation is *venous stasis;* that is, œdema or dropsy of various organs appears. If the left ventricle is especially weakened, dropsy appears first in the legs, on account of the influence of gravity, soon after in the genitals, lungs, liver, and the serous cavities. Engorgement of the lungs is especially marked in cases of mitral disease with weakening of the right ventricle, and is manifested by dyspnœa, cyanosis, cough, and hæmoptysis. In many cases, however, dropsy is very irregularly and unaccountably distributed, and does not follow the rules just given. In pure aortic disease, uncomplicated by leakage of the mitral valve, dropsy is a relatively late symptom, and precordial pain (angina pectoris) is more prominent.

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[1] Rosenbach brings forward evidence to show that the arteries, the lungs, and other organs actively assist in maintaining compensation.

Functional Tests of Compensation.—After a considerable trial of the methods by which it has been proposed to test cardiac power through watching the heart's response to measured "doses" of work, I am convinced that the best tests are the ordinary duties and pleasures of life, which step by step the patient naturally tries in convalescence.

IV. Hypertrophy and Dilatation

Since cardiac hypertrophy or dilatation are not in themselves diseases, but may occur in any disease of the heart (valvular or parietal), it seems best to give some account of them and of the methods by which they may be recognized, before taking up separately the different lesions with which they are associated.

1. *Causes*

1. Vascular hypertension (unknown cause, nephritis, arteriosclerosis).
2. Valvular disease (mostly "rheumatic").
3. Syphilitic aortitis (with aortic regurgitation or aneurism).
4. Adherent pericardium.
5. Moderate hypertrophy and dilatation are often found in Graves' disease, in pernicious anæmia and in leucæmia.

In 1082 cases of hypertrophy and dilatation found in 4000 consecutive necropsies at the Mass. General Hospital, the following possibly causative factors were present:

Arterio-sclerosis	279
Valvular heart disease	79
Nephritis	77
Syphilitic aortitis	24
Chronic pericarditis	24
Pernicious anæmia	8
Leucæmia	6
Hyperthyroidism	3
Unknown cause	210
Total	734

Also the following combinations:

Arterio-sclerosis and nephritis	126
Arterio-sclerosis and chronic valvular endocarditis	36
Arterio-sclerosis and myocarditis	26
Arterio-sclerosis and syphilitic aortitis	17
Chronic endocarditis and pericarditis	11
Nephritis and chronic valvular endocarditis	10
Other combinations	122
Grand Total	1082

I have never seen any evidence that excessive muscular work is *by itself* capable of causing cardiac hypertrophy and dilatation. I have never seen "athlete's heart" in the sense of chronic hypertrophy, or of acute dilatation produced *in the previously sound heart* by violent exertion. It is in the previously diseased heart that athletic "stunts" often produce failure. After a "Marathon run" the heart is of normal size or even smaller than usual.

In valvular disease the greatest degree of hypertrophy is to be seen usually in relatively young persons, and especially when the advance of the lesion is not very rapid.

Hypertrophy of the heart in valvular disease is also influenced by the amount of muscular work done by the patient, by the degree of vascular tension, and by the treatment. In the great majority of cases of hypertrophy, from whatever causes, both sides of the heart are affected, but we may distinguish cases in which one or the other ventricle is *predominantly* affected.

2. *Signs*

(*a*) *Cardiac hypertrophy affecting especially the left ventricle*

(*a*) High systolic blood pressure is the most constant and reliable of all signs of cardiac hypertrophy and is therefore mentioned here.

(*b*) The apex impulse is usually lower than normal, often in the sixth space, occasionally in the seventh or eighth.[1] It is also farther to the left than normal, but far less so than in cases in which the hypertrophy affects especially the right ventricle. The area of visible pulsation is usually increased, and a considerable portion of the chest wall may be seen to move with each systole of the heart, while frequently there is a systolic *retraction* of the interspaces in place of a systolic impulse.

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[1] This is due partly to a stretching of the aorta, produced by the increased weight of the heart.

(*c*) Palpation confirms the results of inspection and shows us also that the apex impulse is unusually deliberate and diffuse as well as powerful ("heaving impulse"). Percussion shows in many cases that the cardiac dulness is more intense and its area increased downward and to a lesser extent toward the left.[1]

(*d*) If we listen in the region of the maximum cardiac impulse, we generally hear an unusually long and low-pitched first sound, which may or may not be of a greater intensity than normal. Often we hear nothing abnormal except a soft systolic murmur. A very loud first sound is much more characteristic of a cardiac weakness of neurosis than of pure hypertrophy of the left ventricle.

The second sound at the apex (the aortic second sound transmitted) is usually much louder and sharper than usual. Auscultation in the aortic area shows that the second sound at that point is loud and ringing in character. Not infrequently the peripheral arteries (the subclavians, brachials, carotids, radials, and femorals) may be seen to pulsate with each systole of the heart. This sign is most frequently observed in cases of hypertrophy of the left ventricle, which are due to aortic regurgitation, but is by no means peculiar to this disease and may be repeatedly observed when the cardiac hypertrophy is due to arterio-sclerosis, Graves' disease, or anæmia.

The radial pulse wave has no constant characteristics, but depends rather upon the cause which has produced the hypertrophy than upon the hypertrophy itself.

The electrocardiogram usually shows characteristic changes.

(*b*) *Cardiac Hypertrophy Affecting Especially the Right Ventricle*

It is much more difficult to be certain of the existence of enlargement of the right ventricle than of the left. Practically we have but three reliable physical signs:

(*a*) Increase in the transverse diameter of the heart, as shown by the position of the apex impulse and by percussion of the left border of the heart; and

(*b*) Accentuation of the pulmonic second sound, which is often palpable as well as audible.

(*c*) A characteristic alteration of the electrocardiogram.

The apex beat is displaced both to the left and downward, *but especially to the left*. In cases of long-standing mitral disease, the cardiac impulse may be felt in mid-axilla, several inches outside the nipple, and yet not lower down than the sixth intercostal space. In a small percentage of cases (*i.e.*, when the right auricle is engorged), an increased area of dulness to the right of the sternum may be demonstrated. Accentuation of the pulmonic second sound is very frequent in hypertrophy of the right ventricle, though it is not constant or peculiar to that condition. It may be heard, for example, in cases of pneumonia when no such hypertrophy is present, but in the majority of cases of cardiac disease we may infer the presence, and to some extent the amount of hypertrophy of the right ventricle from the presence of a greater or lesser accentuation of the pulmonic second sound. The radial pulse shows nothing characteristic of this type of hypertrophy.

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[1] Post mortem enlarged left ventricle is often found despite the absence of the above signs in life because it extends backward out of our reach.

Epigastric pulsation gives us no evidence of the existence of hypertrophy of the right ventricle, despite contrary statements in many text-books. Such pulsation is frequently to be seen in persons with normal hearts, and is frequently absent when the right ventricle is obviously hypertrophied. It is perhaps most often due to an unusually low position of the whole heart.

3. *Dilatation of the Heart*

Dilatation almost never occurs without accompanying hypertrophy. Moreover, it cannot be considered as an unmixed evil. In aortic regurgitation and most other types of cardiac disease it is inseparably linked with hypertrophy and is present long before compensation fails. Probably it is the pathological *fatigue* of a dilated

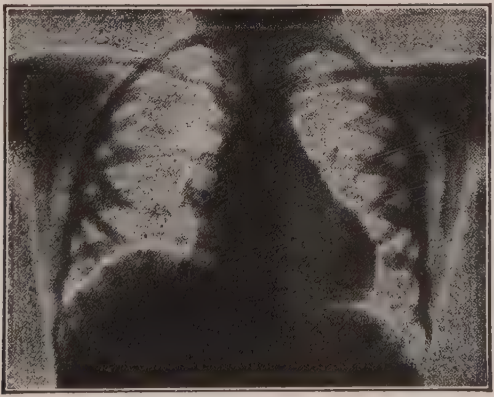

FIG. 132.—Dilated Heart. From v. Ziemssen's Atlas.

and hypertrophied heart that causes stasis and the other abnormalities next described.

(1) *Acute Dilatation.*—Immediately after severe muscular exertion, as, for example, at the finish of a boat race, or of a two-mile run (especially in persons not properly trained), an acute cardiac dilatation has been said to occur. I have never seen this in healthy persons, though Hornung (*Berl. klin. Woch.*, xlv, 1769) believes that with the fluoroscope he has identified cases of acute dilatation in healthy persons after fright, sexual excitement, high altitude, and other strains. In debilitated or poorly nourished subjects, or when the heart has been previously weakened by diseases, such an acute dilatation may be serious or even fatal in its results.

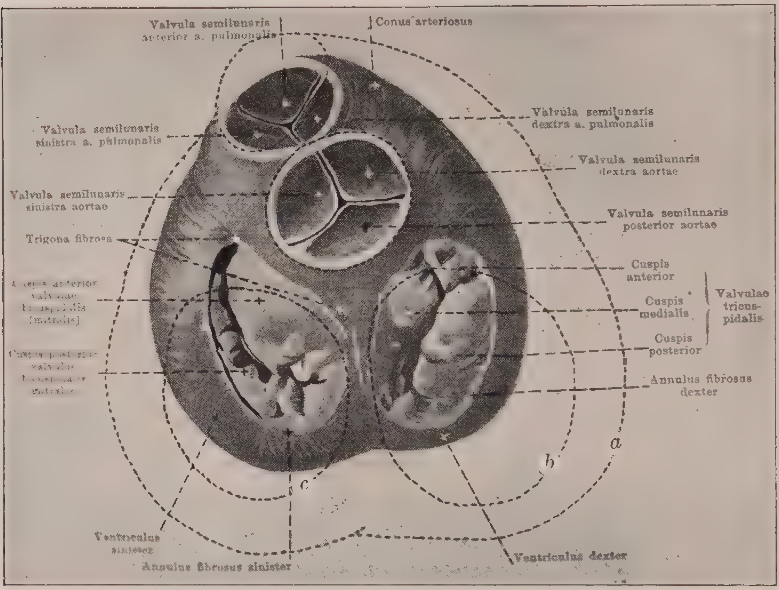
Valvula semilunaris anterior a. pulmonalis
Conus arteriosus
Valvula semilunaris dextra a. pulmonalis
Valvula semilunaris sinistra a. pulmonalis
Valvula semilunaris dextra aortae
Valvula semilunaris sinistra aortae
Valvula semilunaris posterior aortae
Trigona fibrosa
Cuspis anterior
Cuspis anterior valvulae bicuspidalis (mitralis)
Cuspis medialis
Valvulae tricuspidalis
Cuspis posterior
Cuspis posterior valvulae bicuspidalis (mitralis)
Annulus fibrosus dexter
a
b
c
Ventriculus sinister
Annulus fibrosus sinister
Ventriculus dexter

FIG. 133.—The Base of the Contracted Heart Showing Sphincteric Action of the Muscular Fibres Surrounding the Mitral and Tricuspid Valves. The outer dotted line is the outline of the relaxed heart. The inner dotted circles show the size of the mitral and tricuspid valves during diastole. *a*, Outline of the heart when relaxed; *b*, outline of the relaxed tricuspid valve; *c*, outline of the mitral orifice during diastole. (After Spalteholz.)

(2) *Chronic dilatation* with hypertrophy comes as a result of valvular disease or other causes, and gives rise to the same physical signs as those of acute dilatation, from which it differs chiefly as regards the accompanying physical phenomena and the prognosis.

The mitral and tricuspid orifices are closed not simply by the shutting of their valves, but also in part by the sphincter-like action of the circular fibres of the heart wall (see Fig. 133) and the contraction of the papillary muscles (Fig. 134).

In birds, the tricuspid orifice has no valve and is closed wholly by the muscular sphincter of the heart wall.

In conditions of very acute cardiac failure, such as may occur after a hard run, the papillary muscles are in all probability relaxed.

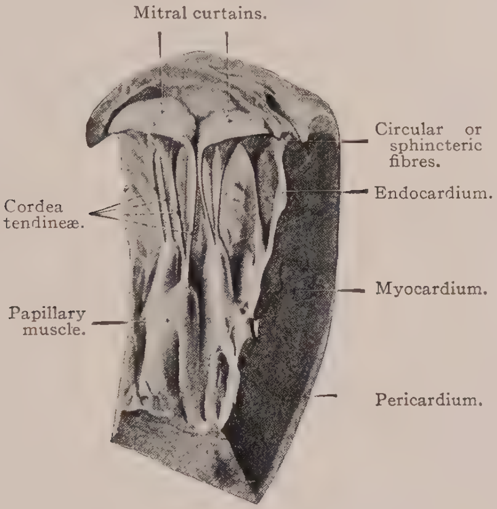

FIG. 134.—The Mitral Valve Closed, Showing the Action of the Papillary Muscles. (After Spalteholz.)

Briefly stated, the signs of predominant dilatation of the heart. whether acute or chronic, are:

(*a*) *Feebleness and irregularity* of the apex impulse and of the radial impulse; (*b*) *enlargement* of the heart, as indicated by inspection, palpation, and percussion, and (sometimes) (*c*) widely spread systolic *murmurs;* (*d*) often gallop rhythm (see above, p. 180); (*e*) evidence of stasis at the periphery.

(*a*) *Predominant Dilatation of the Left Ventricle*

Inspection shows little that is not better brought out by palpation. Palpation reveals a "flapping" cardiac impulse, or a vague shock displaced both downward and to the left and diffused over an abnormally large area of the chest wall. Percussion verifies the position of the cardiac impulse.

On auscultation, the first sound is usually *very short and sharp*, but *not feeble* unless it is accompanied by a murmur. A systolic murmur is sometimes heard at the apex of the heart. The aortic second sound, as heard in the aortic area and at the apex, is feeble. Peripheral blood-pressure sometimes falls.

(*b*) *Predominant Dilatation of the Right Ventricle*

The failure of muscular tone is shown in extreme cases by an increase in the area of cardiac dulness to the right of the sternum (corresponding to the position of the *right auricle*), by feebleness of the pulmonic second sound and especially by cyanosis and signs of congestion and engorgement of the lungs. When this latter event occurs, one may have also systolic pulsation in the jugular veins, and rarely in the liver.

The diagnosis of dilatation of the heart seldom rests entirely upon physical signs referable to the heart itself. In acute cases our diagnosis is materially aided by a knowledge of the cause, which is often tolerably obvious. In chronic cases the best evidence of dilatation is often that furnished by the venous stasis which results from it.

In conditions of profound nervous debility, excitement, or exhaustion, weakening of the cardiac muscles is often associated with systolic murmurs, which cease with the removal of their cause. Stress has been laid upon these points by Da Costa, by Prince, and recently in the World War.

Systolic murmurs at the base and at the apex are often heard in aortic regurgitation with the dilatation of the left ventricle which that lesion produces, also in chronic nephritis and arterio-sclerosis.

The *results* of any form of mitral disease occur in this order.

(1) *Dilatation and hypertrophy of the left auricle*, which has to receive blood from the lungs but cannot empty itself freely into the left ventricle.

(2) The overfilled left auricle cannot receive the blood from the lungs as readily as it should; hence the blood "backs up" in the lungs and thereby increases the work which the right ventricle must do in order to force the blood through them. Thus result œdema of the lungs, and—

(3) *Hypertrophy and dilatation* of the right ventricle, which in turn becomes sooner or later overcrowded so that tricuspid leakage occurs.

(4) The muscular capacity possessed by the right auricle is soon exhausted, and we get then—

(5) *General venous stasis*, which shows itself first as systolic pulsation in the jugulars and in the liver, and later in the tissues drained by the portal and peripheral veins. This venous stasis increases the work of the left ventricle, and so we get—

(6) *Hypertrophy and dilatation of the left ventricle.* Hypertrophy of the left ventricle is also produced by the increased work necessary to maintain sphincter action at the mitral orifice.

At last the circle is complete. Every chamber in the heart is enlarged and fatigued. Failure is imminent.

CHAPTER XII

THE CLASSIFICATION OF CARDIO-VASCULAR DISEASES

I have found it convenient to make four groups out of those whose illness appears to be due chiefly to circulatory weakness. The classification which appears below is based on the figures which accompany it, and on the last ten years' experience with diseases of the circulation in the wards and *post-mortem* room of the Massachusetts General Hospital.

The tentative nomenclature and relative frequency of the different types is as follows:

		Occurrence in 600 clinical cases, per cent.	Occurrence in 126 cases post mortem, per cent.
1	"Rheumatic"	46	42
2	Syphilitic	12	13
3	Hypertensive (arteriosclerotic and nephritic)	34	40
4	Congenital malformations	0	1
	Doubtful cases[1]	8	4

These two sets of figures do not overlap. The clinical cases were all studied between 1910 and 1914. Those autopsied had died between 1896 and 1905. The two series were worked out at different times and without any comparison of the figures.

1. The terms need some definition: "*Rheumatic*" here includes the cases of weakened myocardium, acute pericarditis, adherent pericardium, and valvular disease associated with:

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[1] One-fifth of these are goitre hearts.

(*a*) Acute polyarthritis (non-gonorrhœal).
(*b*) Acute chorea (Sydenham's type).
(*c*) Acute septic tonsillitis.
(*d*) Primary endocarditis.

Only cases with negative Wassermann are included. Sixty per cent. of the cases begin before the twenty-second year, and 60 per cent. are in girls. It seems probable but not proved that some type of streptococcus is the cause in all cases of this type. The proved gonorrhœal cases are so few that I have left them out of account. The scarlatinal cases are probably streptococcic.

Cases of malignant or ulcerative endocarditis and Libman's "subacute and chronic bacterial endocarditis" are here included in the same group.

Ultimately the word "rheumatic" may well be changed to streptococcic.

2. *Syphilitic heart disease* is here used to include syphilitic aortitis, (with and without aneurism), and all the weakened hearts associated *with proved syphilis and without other obvious cause.* Seventy per cent. of the cases are in middle-aged men.

3. *Hypertensive Heart Disease, Arteriosclerotic Type.*—(*a*) The average age in my series is 59 years. The arteries show extensive changes. No evidence of syphilis or of the valve lesions ordinarily associated with "rheumatism" was discovered. I leave unanswered the question whether arteriosclerosis is a result or a cause of hypertension, though I incline to believe that the two are independent.

(*b*) *Nephritic* (*or Nephrogenous*) *Type.*—Average age 36. Obvious nephritis of the glomerular or vascular type. Negative Wassermann. No valve lesions.

The *congenital malformations* need no further explanation here. Other less defined types will be mentioned later (see page 275).

I. RHEUMATIC HEART DISEASE

Under this heading I shall discuss:

(*a*) Acute endocarditis.
(*b*) Pancarditis (especially in children).
(*c*) Mitral disease.
(*d*) Aortic disease.
(*e*) Disease of the tricuspid and pulmonary valves.

The "rheumatic" type of myocarditis cannot, I believe, be recognized clinically and will not, therefore, be separately discussed. It will be referred to in connection with the discussion of *Compensation*.

1. *Acute Endocarditis*

There are no positive or characteristic physical signs of this form of the disease. We suspect it by reasoning from one member to another within a familiar pathologic group. When fever and leucocytosis without any other known cause are associated with acute arthritis or chorea, or persist unexplained after an acute tonsillitis has subsided, one may conjecture that acute endocarditis is present, *i.e.*, soft, friable vegetations on the heart valves. The conjecture approaches knowledge if we find one or more cardiac murmurs which change in time, position, quality or intensity from day to day, and is confirmed if evidence of embolism appears in the subcutaneous tissues, in the kidney, spleen, brain, eye, or peripheral arteries.

The diagnosis is seldom made without the presence of chronic "rheumatic" lesions in the heart, on some of which the acute process is often grafted.

It is often present as a terminal lesion superimposed on chronic valvular defect, less often in chronic wasting diseases. It is sometimes associated with a septic focus such as peritonitis.

2. *Rheumatic Pancarditis of Children*

The rheumatic (*i.e.*, streptococcic) infections of children and young adults often attack simultaneously the endocardium, myocardium, and pericardium.

It may be impossible to determine in a given case whether the infection has attacked one, two, or all three of these parts. Single, double, or triple invasion may produce identical signs, *i.e.*, *rapid, perhaps irregular heart action, a systolic murmur usually loudest in the apex region, and as time goes on more or less extension of the heart's borders*. The evidence of acute pericarditis (see below, p. 238) may not be recognizable. If the aortic valve is affected we can generally make that out, but if the mitral alone is involved there are no signs in the early acute stages of the disease by which we can distinguish the systolic murmur of mitral vegetations from the similar murmur produced by acute rheumatic infection with or without pericarditis.

Years ago I used to call these cases "mitral regurgitation" and this is still their popular title, but I have ceased to use it since I have watched the disease get well and heard the murmur more and more faintly till it disappeared altogether. In such a case there *may* have been a true mitral regurgitation due to relaxation of the mitral sphincter from weakening of the circular muscle fibres about the valve orifice. But without *post-mortem* this is conjecture only and though the necropsy often does *not* verify it we cannot deny that during life there may have been atony not demonstrable in the dead tissue.

It will now describe separately the physical signs which tend to convince us that one or another element within the circulatory apparatus is especially affected by the "rheumatic" infection.

3. *Mitral Endocarditis (Rheumatic Type)*

(*a*) *Early Stages*

In the beginning of a rheumatic heart trouble which the latter evidence proves to be "*mitral*" (*i.e.*, more severe there than elsewhere), we usually have no signs except a *systolic murmur* loudest at or near the apex of the heart.

(*b*) *Later Stages*

(*a*) As time goes on *hypertrophy and dilatation* of the heart gradually develop. It is customary to speak of hypertrophy as an early conservative or *compensatory* process, and of dilatation as a later more or less accidental and harmful result. But we usually find the two processes combined and have no good reason to call one useful and the other harmful. Both sides of the heart increase in thickness and in capacity. *Predominant* hypertrophy and dilatation of the right side is, however, the rule in mitral disease.

The physical evidence of enlargement is shown not to the right of the sternum (where the enlarged right auricle might appear), not in the epigastrium (where the right ventricle presents), but on the left cardiac border.

The heart's apex is found normally outside the nipple line in children and as a rule is in the fourth interspace until the child reaches the 9th, 10th, or 11th year. About that time, or slightly later, it is found in the fifth space and in the nipple line—later within it.

Beyond these limits and especially to the left, the heart of early mitral endocarditis gradually extends until it is recognized by palpation. Percussion is rarely needed in examining the thin flexible chests of children and young adults in whom mitral disease usually begins.

The left auricle also thickens and stretches but presents no physical signs.[1]

(b) *Accentuation of the Pulmonic Second Sound.* As a rule there is no considerable change in the second sound until mitral endocarditis reaches its later or *stenotic* form. But in a minority of cases the second sound on the left side of the sternum becomes louder and sharper than normal even in the earlier stages of mitral disease.

At this prestenotic stage of mitral disease, after the early acute infection (with fever, leucocytosis, and anæmia) has passed, and before the mitral valve has become notably contracted, we have then two physical signs which are fairly constant and one that is inconstant.

1. Systolic murmur at the apex.
2. Slight enlargement of the heart, especially to the left.
3. (In a minority of cases) accentuation of the pulmonic second sound.

These three signs appear in the order just given. For months there may be no abnormality except a systolic murmur indistinguishable from those which pass away and do not develop into stenotic mitral disease.

If the murmur persists it is often followed in a few months or years by an extension of the apex toward the left, by accentuation of the pulmonic second sound and finally by evidence of stenosis.

The Murmur.—In children the murmur of early mitral disease may be among the loudest of all murmurs to be heard in valvular disease, but this does not necessarily imply that the lesion is a very severe one. A murmur which *grows louder* under observation in a well-compensated valvular lesion may mean an advance of the disease, but if the case is first seen after compensation has failed, a faint, variable whiff in the mitral area may mean the severest type of lesion. As the patient improves under the influence of rest and cardiac tonics, such a murmur may grow very much louder, or a murmur previously inaudible may appear.

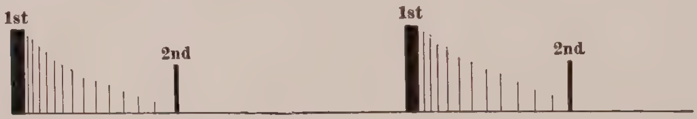

FIG. 135.—Diagram to Represent Systolic Mitral Murmur. The heavy lines represent the normal cardiac sounds and the light lines the murmur, which in this case does not replace the first sound and "tapers" off characteristically at the end.

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[1] Except by x-ray or by the electro-cardiogram (*vide infra*).

The *length* of the murmur varies in different cases, but as a rule it is longer than those which are "functional" in origin. It begins abruptly but usually "tails off" at the end of systole (see Fig. 135). The first sound of the heart may or may not be replaced by the murmur (see Fig. 136). When the sound persists and is heard either with or before the murmur, one can infer that the lesion is relatively slight in comparison with cases in which the first sound is wholly obliterated. *Post-systolic* or *late systolic* murmurs, which are occasionally heard in early mitral disease, are said to point to a relatively slight amount of disease in the valve (see Fig. 137).

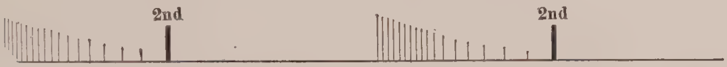

FIG. 136.—Systolic Mitral Murmur Replacing the First Sound of the Heart.

When compensation fails, the murmur may altogether disappear for a time, and if the patient is then seen for the first time and dies without rallying under treatment, it may be impossible to make the diagnosis.

The murmur of early mitral disease is loud and hence is conducted in all directions, but especially toward the axilla and to the back. In the latter situation it is usually louder than it is in mid-axilla, and occasionally it is heard as loudly in the back as anywhere else in the chest.

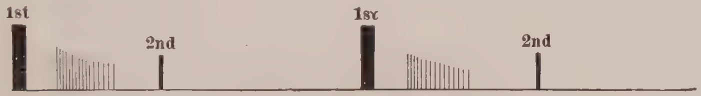

FIG. 137.—Late Systolic Murmur. The first sound is clear and an interval intervenes between it and the murmur.

If accentuation of the pulmonic second sound is present, we recognize it not because it is louder than the aortic second sound, since this is true in the vast majority of cases in healthy individuals under thirty years of age. Pathological accentuation of the pulmonic second sound means *a greater intensity of the sound than we have a right to expect at the age of the individual in question.* Occasionally the pulmonic second sound is loudly reduplicated, but as a rule this points to advanced stenosis of the mitral valve, *i.e.*, to the later stages of mitral endocarditis.

In children (in whom adhesive pericarditis often complicates the disease) a systolic THRILL may not infrequently be felt at the apex, and the precordia may bulge.

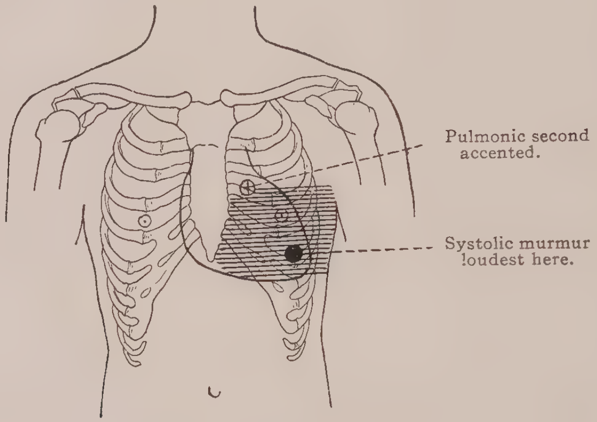

FIG. 138.—Early Mitral Disease. The murmur is heard over the shaded area as well as in the back.

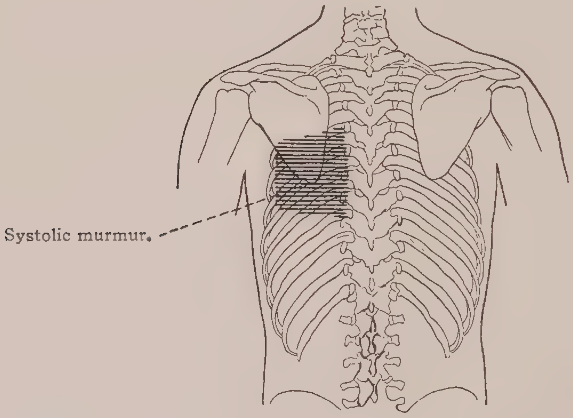

FIG. 139.—Early Mitral Disease. Murmur heard over the shaded area.

The pulse usually shows nothing characteristic at this stage of the disease, although it may be weak when compared to the powerful beat at the apex, in case the stenotic element in the lesion is increasing rapidly. But arrhythmia at this period is much less common than in cases advanced to the stage of stenosis.

Differential Diagnosis

The murmur of early mitral disease may be confused with:

(1). Tricuspid regurgitation.

(2) Functional murmurs.

(3) Stenosis or roughening of the aorta or aortic valves.

(1) The post-mortem records of the Massachusetts General Hospital show that in the presence of a murmur due to mitral disease it is very easy to fail altogether to recognize a tricuspid regurgitant murmur. Only 5 out of 29 cases of tricuspid stenosis and regurgitation found at autopsy were recognized during life. Sir Clifford Allbutt's figures from Guy's Hospital are closely similar. In the majority of these cases, "mitral regurgitation" was the diagnosis on which attention was concentrated during the patient's life. This is all the more excusable because the tricuspid area is so wide and uncertain. Murmurs produced at the tricuspid orifice are sometimes heard with maximum intensity just inside the apex impulse, and if we have *also* an apical murmur, it may be impossible under such circumstances to distinguish it from the tricuspid murmur. Sometimes the two are of different pitch, but in most cases tricuspid regurgitation must be recognized *indirectly* if at all, *i.e.*, through the evidence given by venous pulsation in the jugular veins and in the liver, and through the rapid accumulation of ascites and œdema of the legs.

(2) "Functional" murmurs are usually systolic and may have their maximum intensity at the apex of the heart, but in the great majority of cases they are heard best over the pulmonic valve or just inside or outside the apex beat (Potain). They are faint or inaudible at the end of expiration, and are more influenced by position than organic murmurs are. In the upright position they are often very faint. They are usually short, are rarely transmitted beyond the precordia and are unaccompanied by any evidences of enlargement of the heart, by any pathological accentuation of the pulmonic second sound,[1] or any evidences of engorgement of the lungs or general venous system.

Cardio-respiratory murmurs are usually systolic, and as they are often heard in and about the mitral area they are frequently mistaken for evidence of a mitral lesion. From this they should be distinguished by their variation or cessation in certain phases of respiration and by the absence of any other evidence of valvular disease.

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[1] It must be remembered that in chlorosis, a disease in which functional murmurs are especially prone to occur, the pulmonic second sound is often surprisingly loud, owing to a retraction of the left lung, which uncovers the root of the pulmonic artery.

(3) Roughening or narrowing of the aortic valves or of the aortic arch may produce a systolic murmur with maximum intensity in the second right intercostal space, but this murmur is not infrequently heard all over the precordia and most plainly at the apex, so that it may simulate the murmur of early mitral lesions. The aortic murmur may indeed be heard more plainly at or just inside the apex. In such cases diagnosis may be impossible, as there may be enlargement of the heart as well. Accentuation of the pulmonic second sound is not constant enough in early (prestenotic) mitral disease to constitute a differential point of much importance.

The Question of "Mitral Regurgitation" in Early Rheumatic Mitral Disease

Throughout this section on the early or prestetonic stages of mitral disease I have refrained from using the classical term "*mitral regurgitation*." I have done so because I know no means by which we can recognize with any certainty, either *ante-mortem* or *post-mortem*, that such regurgitation is, or has been, a fact. That there may be regurgitation through the mitral at any period of the disease is entirely possible. Early coxcomb vegetations *may* hold the valves apart so that they cannot close. Relaxation of the papillary muscles or shortening of tendinous cords *may* well permit a reflux. Muscular weakness (with or without a demonstrable myocarditis) *may* weaken the sphincteric action of the circular fibers about the base of the leaflets so that they do not fill up the mitral orifice.

Any or all of these events are possible, but we have (so far as I know) no way of being even approximately sure that in a given case they are happening. On the other hand, we do know that people often die with the signs of "mitral regurgitation" when *post-mortem* gives no evidence either of anatomical change in the valves or of an increase of the valve circumference.

Add to this that, when we *do* find *post-mortem* an increase in the valve circumference, the *ante-mortem* findings are often widely different from those traditionally associated with "*mitral regurgitation*."

So far as I have been able to observe *post-mortem* the "rheumatic" (streptococcic) lesions of the mitral valve are almost never such as one could reasonably expect to produce *regurgitation alone*, *i.e.*, without stenosis as well. One finds vegetations or roughenings such as might well produce a murmur without any regurgitation. One finds *at this stage* very little predominance of right ventricular hypertrophy.

Later one finds the valves so stiffened and adherent that regurgitation as well as obstruction is practically certain. But pure regurgitation—before stenosis has occurred—and in the early stages of rheumatic mitral disease, is—so far as I can see—a very rare possibility—no more.

Of its occurrence, or possible occurrence in other types of cardiac disease (syphilitic, arteriosclerotic, or nephritic) I shall speak during the discussion of those diseases.

The practical importance of these considerations is, I think, considerable. Boys with a loud systolic murmur at the apex are frequently refused permission to enter athletic contests or army life. Older men are refused life insurance. This is often wholly wrong. *Such murmurs are no evidence of disease.* I have known boys row a four-mile boat race or fight through a war with such a murmur and be none the worse for it. It often disappears later in life.

For practical purposes *we should disregard systolic murmurs provided the response to exercise* is satisfactory, the cardiac sounds, dimensions and contractions otherwise negative, and a history of rheumatic disease absent.

But it must be remembered that in cases of early mitral stenosis the presystolic murmur is often inaudible and only a systolic evident, unless we stir up the heart by exercise or amyl nitrite.

Mitral regurgitation should never be the only or the primary diagnosis made. We may rightly diagnose :

1. *Mitral stenosis* and regurgitation.
2. *Arteriosclerosis* with cardiac hypertrophy and dilatation (and possibly relative mitral regurgitation).
3. *Chronic nephritis* or pericarditis with the same results.
4. *Hyperthyroidism* with the same results.
5. *Syphilitic aortitis* with the same results.
6. *Acute rheumatic* (streptococcic) *myocarditis* with the same results.

But in all these cases the mitral regurgitation, if it occurs, is a minor result (like congested lungs), never a primary cause. Primary mitral regurgitation is the commonest wrong diagnosis in the field of the circulation and does the greatest harm to the patient, especially when it excludes him from life insurance, from athletics, or from war.

(*c*) *Late Mitral Disease—Stenosis*

In children we often see acute mitral endocarditis with a systolic murmur at the apex. But unless they have had previous acute attacks so that the total duration of the disease is a year or more, we do not often get the physical signs presently to be described as those of stenosis. Much more often we find the typical signs of stenosis in young adults (especially women) or in older persons, who have a history of rheumatic arthritis, chorea, or tonsillitis years before.

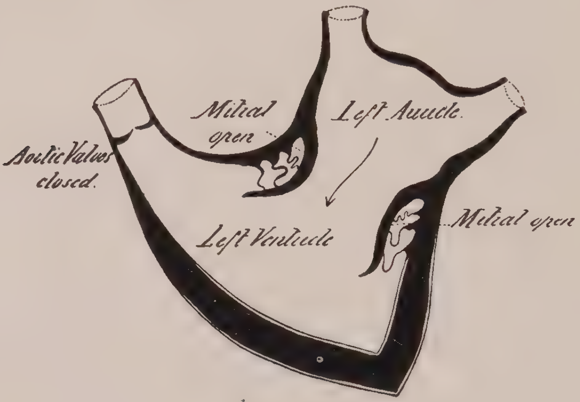

FIG. 140.—Diagram to Represent the Position of the Valves in the Normal Heart during Diastole, the Open Mitral Allowing the Blood to Flow from the Left Auricle, the Aortic Closed.

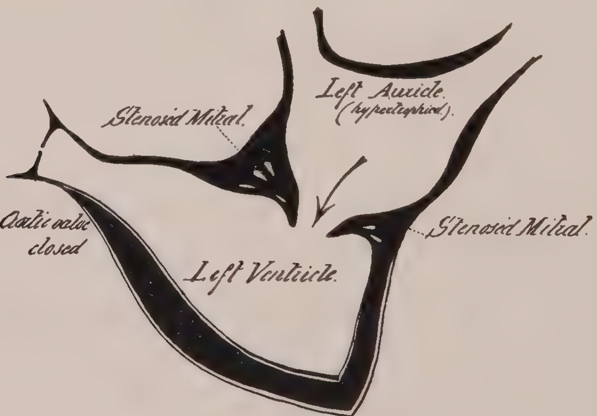

FIG. 141.—Mitral Stenosis—Period of Diastole. The blood flowing from the left auricle is obstructed by the thickened and adherent mitral curtains.

Since "rheumatic" arthritis and chorea are commoner in girls than in boys, it is natural that the commonest late cardiac result of these infections (mitral stenosis) should be commoner in women. Not infrequently we find advanced stenosis in persons who have no remembrance of any previous rheumatism, chorea, tonsillitis, or other form of streptococcus infection. But I find no good evidence that any of the other assigned causes (such as tuberculosis) plays any causative rôle in mitral disease.

Post-mortem mitral stenosis is not infrequently the obvious and fundamental cause of death. It proved such in 104 of 4000 autopsies at the Massachusetts General Hospital. On the other hand there was but one case (No. 2825) in which mitral regurgitation seemed the cause of death. When an enlarged mitral orifice was demonstrated, it was usually as a minor feature—death being due to typhoid fever. to arteriosclerosis, or some such cause lying deeper than the stretched orifice.

In typical cases of stenosis the valves are fused together and stiffened to form an irregular cone projecting into the ventricle from the direction of auricle (see Figs. 142 and 143).

The orifice is never round but presents an irregular slit (fish mouth or button-hole) which may admit one finger (instead of three as normally) or may barely admit a probe. It is amazing to see such a narrowing in the heart of a person who until a few days before death has done active work with but little inconvenience.

Physical Signs

Mitral stenosis may probably exist for years without producing any physical signs by which it may be recognized and even after signs have begun to show themselves they are more fleeting and inconstant than in any other valvular lesion of the heart. In the early stages of the disease the heart may appear to be entirely normal if the patient is at rest, and especially if examined in the recumbent position, characteristic signs being elicited only by exertion. Or again a presystolic roll which is audible with the patient in the upright position may disappear in the recumbent position; or a murmur may be heard at one visit, at the next it may be impossible to elicit it by any manœuvre, while at the third visit it may be easily heard again. These characteristics explain to a certain extent the fact that differences of opinion so often arise regarding the diagnosis of mitral stenosis, and that out of 130 cases in which this lesion was found at autopsy at the Massachussetts General Hospital, only 73 or 56 per cent. were recognized during life. No common lesion has been so frequently overlooked in our records.

In the earliest stage at which the stenosis can be recognized with certainty, *there are no abnormal signs* when the patient is at rest *except a systolic murmur.* This murmur often has a peculiar "explosive" or hissing quality like a jet of steam escaping suddenly and under pressure. Its earliest portion is strongly accented like a sforzando in music. It is short, high pitched, and dies away rapidly.

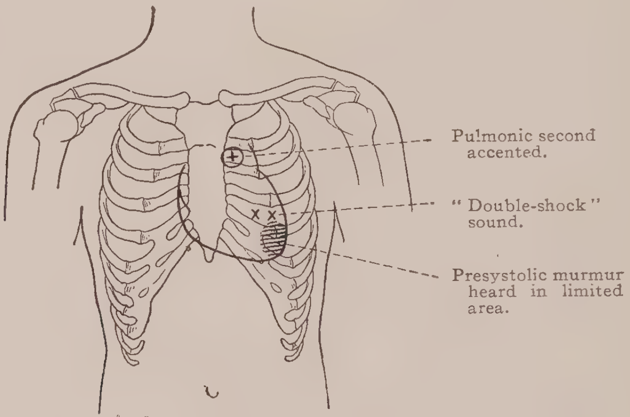
Pulmonic second accented.
"Double-shock" sound.
Presystolic murmur heard in limited area.

FIG. 142.—Mitral Stenosis.

Now if by exertion or by the action of amyl nitrite, the heart's contraction is invigorated a low-pitched *presystolic* role will appear faintly, then distinctly, rapidly disappearing again as the heart quiets down. With the increase of cardiac activity under amyl nitrite I have also seen the presystolic thrill and the double shock sound (presently to be mentioned) appear for a few beats and then vanish.

As a rule the signs just mentioned are those of the very earliest stages of the disease; but in some cases they are probably the only ones present during the observation period. At any rate no presystolic murmur was heard at any time in 19 out of 59 cases of pure mitral stenosis autopsied at the Massachusetts General Hospital but in most of these a systolic murmur was audible. For the most part it is probably true that a systolic explosive murmur is the only one to be heard in the earliest stages of the stenotic lesion.

From this point on the symptoms may be divided into three stages according to the extent of progress in the case.

I

In the first stage palpation shows that the apex beat is little if at all displaced, and percussion reveals little if any increase in the area of cardiac dulness; there is often local tenderness to be elicited near the apex. If one lays the hand lightly over the origin of the apex beat, one can generally feel the *purring presystolic thrill* which is so characteristic of this disease. This thrill is more marked in the second stage of the disease, but can generally be appreciated even in the first. It runs up to and ceases abruptly with the very *sharp first sound*, the sudden *shock* of which may be appreciated even by palpation. On auscultation one hears, especially after the patient has been exerting himself, and particularly if he leans forward and to the left, a *short low-pitched rumble or roll immediately preceding the systole.* The murmur *seems* to be a *crescendo*, growing louder as it approaches the first sound, but phonocardiograms do not show any such crescendo. At this stage of the disease the second sound can still be heard at the apex. The first sound is often sharply accented or snapping, and communicates a very decided shock to the ear. As a rule, the murmur is closely confined to the region of the apex beat and not transmitted any considerable distance in any direction. I have seen cases in which it was to be heard only over an area the size of a half-dollar.[1] *The pulmonic second sound may be accented* and is often loudly reduplicated ("*double-shock sound*"—Sansom) at this stage of the disease, but this doubling is more frequent later.[2]

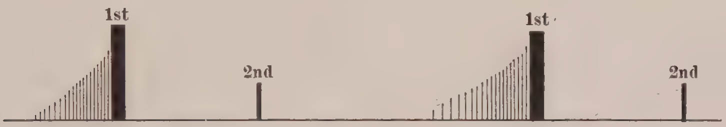

FIG. 143.—The Murmur of Mitral Stenosis—First Stage. The place of the murmur and its crescendo character indicated by the position of the light lines just before the first sound and by their increasing length.

Arrhythmia is not infrequently present even in the early stages of the affection. The heart may be regular while the patient is at rest, but slight exertion is often sufficient to produce marked irregularity.

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[1] It may, however, be widely transmitted to the left axilla and audible in the back or even over the whole precordia.

[2] This is the opinion of most observers. Sansom has stated that the "double-shock sound" may precede *all* other evidences of mitral stenosis.

II

In the *second stage* the murmur and thrill are usually longer and may occupy the whole of diastole, beginning with considerable intensity just after the reduplicated second sound, quickly diminishing until barely audible, and then again increasing with a rapid crescendo up to the first sound of the next cycle.[1] These changes may be graphically represented as in Figs. 144 and 145. Diastole and the A-s–V-s interval of the phlebogram (see above, p. 114) are now still more prolonged, so that the characteristic rhythm of this lesion is even more marked than in the earlier stages of the disease. In many cases at this stage no second sound is to be heard at all at the apex, although at the pulmonic orifice it is loud and almost invariably double. (This is one of the reasons for believing that the second sound which we usually hear at the apex is the transmitted aortic second sound. In mitral disease the aortic valves shut feebly owing to the relatively small amount of blood that is thrown into the aorta.)

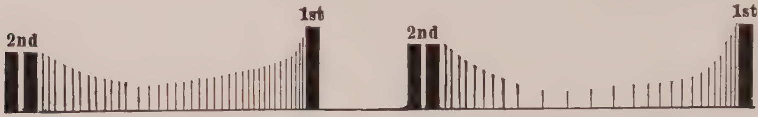

FIG. 144.—Type of Presystolic Murmur Often Heard in the Second Stage of Mitral Stenosis. Here the murmur fills the whole of diastole, with a gradual increase of intensity as it approaches the first sound. No second sound is audible at the apex.

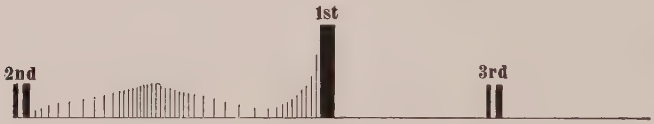

FIG. 145.—Type of Presystolic Murmur Sometimes Heard in the Second Stage of Mitral Stenosis. There is a double crescendo. The second sound seems reduplicated.

At this stage of the disease enlargement of the heart begins to make itself obvious. The apex impulse is displaced to the left—sometimes as far as the mid-axillary line, and often descends to the sixth interspace.

The instability and fleeting character of the murmur in the earlier stages of the disease are much less marked in this, the second stage. Some murmurs, presystolic or early-diastolic or mid-diastolic, can usually be heard with every beat, but the murmurs vary from instant to instant, they can often be heard all over the precordia. The first sound at the apex still retains its sharp, thumping quality, and is often audible *without the murmur* in the back.

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[1] Often one finds a crescendo in the middle of a long presystolic roll with a diminuendo as it approaches the first sound.

The irregularity of the heart is generally greater at this stage than in the earlier one, and often becomes "absolute" (auricular fibrillation).

III

The third stage of the affection is marked by the weakening or disappearance of the characteristic murmur, and is generally synchronous with the development of failing compensation. The right ventricle becomes dilated sometimes very markedly. Indeed, it may produce a visible pulsating tumor below the left costal border and be mistaken for cardiac aneurism (Osler). The snapping first sound and the "double-shock" sound usually remain audible, but the latter may be absent altogether. Diagnosis in this stage rests largely upon the peculiar snapping character of the first sound, together with the prolongation of diastole and the absolute irregularity of the heart, both in force and rhythm. But usually a faint early diastolic murmur can still be heard along the left sternal border continuously. It is soft and high pitched, not at all suggesting the deep rolling presystolic of earlier stages.

As the disease advances, the irregularity of the pulse becomes more and more marked, and sometimes presents an amazing contrast with the relatively good general condition of the circulation. Even when not more than a third of the beats reach the wrist, the patient may be able to attend to light work and feel very well. Such cases make us feel as if a functioning auricle and a palpable pulse were luxuries rather than necessities.

Under the influence of digitalis the pulse beats are especially apt to assume the *bigeminal* or coupled type. (See above, p. 123.)

Mitral stenosis is in the great majority of cases combined with a systolic mitral murmur, and it often happens that this murmur is so much more prominent than anything suggesting stenosis that the latter escapes observation altogether, especially in the third stage of the disease, when the typical presystolic roll has disappeared. In such cases combined stenosis and regurgitation is to be distinguished by the sharpness of the first sound. The presence of reduplicated second sound, a "double-shock sound" at the outset of the prolonged diastolic pause, and the absolute irregularity of the pulse are further suggestive of mitral stenosis. Moreover, it should be remembered that a death from mitral regurgitation is almost unknown and that *if the patient has a chronic serious mitral disease of the rheumatic type, stenosis is almost certainly present whatever the murmur.*

Mitral stenosis is apt to be associated with hæmoptysis, with engorgement of the liver and ascites, and especially with arterial embolism. *No other valve lesion is so frequently found associated with embolism.* This is owing to the very frequent formation of a "ball" or pedunculated thrombus in the left auricular appendage. Sudden death may result from the impaction of this "ball" in the funnel-like cone of the stenosed mitral orifice. More often bits of the thrombus break loose and are "heard from" in the brain (hemiplegia). In the spleen and kidneys they are usually "silent," but may cause sudden and severe pain. Mesenteric embolism or embolic gangrene of a leg are not uncommon and, like hemiplegia, may be the first symptom of the disease mitral stenosis.

Differential Diagnosis

Other murmurs which may be mistaken for the murmur of mitral stenosis are:

(*a*) The Austin Flint murmur.

(*b*) The murmur of tricuspid stenosis.

(*a*) In 1862 Austin Flint studied two cases in which during life a typical presystolic roll was audible at the apex of the heart, yet in which post mortem the mitral valve proved to be perfectly normal, and the only lesion present was aortic insufficiency. This observation has since been verified by Osler and other observers. I have had many such cases with autopsy. Yet, despite repeated confirmation, Flint's observation still remains unknown to physicians at large. Its importance is this: Given a case of aortic regurgitation, or any other cause producing marked hypertrophy and dilatation of the left ventricle—a presystolic murmur at the apex does not necessarily mean stenosis of the mitral valve even though the murmur has the typical rolling quality and is accompanied by a palpable thrill. It may be only one of the by-effects of the aortic incompetency. How it is that a presystolic murmur can be produced at the apex in cases with big left ventricle, has been much debated, but remains unknown.

Between the "Austin Flint murmur" thus defined and the murmur of mitral stenosis, complicating left-sided hypertrophy, diagnosis may be impossible. If there is no dilatation of the mitral orifice, and no regurgitation, any evidence of engorgement of the pulmonary circuit (accentuation of the pulmonic second sound, œdema of the lungs, hæmoptysis, and cough) speaks in favor of an actual narrowing of the mitral valve, while the absence of such signs and the presence of a Corrigan pulse, or of predominating hypertrophy of the left ventricle tend to convince us that the murmur is of the type described by Austin Flint, *i.e.*, that it does not point to any stenosis of the mitral valve. The sharp, snapping first sound, the thrill and systolic shock so characteristic of mitral stenosis are apt to be modified or absent in connection with murmurs of the Austin Flint type. A positive Wassermann reaction with aortic regurgitation usually means syphilitic aortitis and no mitral stenosis—since the latter is a rheumatic, not a syphilitic, lesion. Hence a presystolic murmur heard in a case showing good evidence of syphilis and of aortic regurgitation but not rheumatic history is probably of the "Flint" type.

(*b*) Tricuspid obstruction.

Luckily for us as diagnosticians, stenosis of the tricuspid valve is a rare lesion. Like mitral stenosis it is manifested by a presystolic rolling murmur whose point of maximum intensity is sometimes over the traditional tricuspid area, but may be at a point so near the mitral area as to be easily confused with stenosis of the later valve.

The difficulty of distinguishing tricuspid stenosis from mitral stenosis is further increased by the fact that the two lesions are both of "rheumatic" origin and almost invariably occur in conjunction. Hence we have two presystolic murmurs, perhaps with slightly different points of maximum intensity and possibly with a difference in quality, but often quite undistinguishable from each other. In the vast majority of cases, therefore, tricuspid stenosis is first recognized at the autopsy, and the diagnosis is at best a very difficult one. When a mitral stenosis seems to yield to treatment less readily and the heart is larger than we should expect, considering the general condition of the patient, we may guess that a tricuspid obstruction (perhaps also an aortic stenosis) is present as well.

Broadbent, Rosenbach, and others have noticed in children who have just passed through an attack of pericarditis a rumbling murmur near the apex of the heart, which suggests the murmur of mitral stenosis. It is often distinguished from this, however, by the absence of any accentuation of the first sound at the apex, as well as by the conditions of its occurrence and by its transiency. Such cases are important, since their prognosis is much less favorable than that of mitral stenosis.

Phear (*Lancet*, September 21, 1895) investigated 46 cases in which a presystolic murmur was observed during life and no mitral lesion found at autopsy. In 17 of these there was *aortic regurgitation at* autopsy. In 20 of these there was *adherent pericardium* at autopsy, *in* 9 *nothing more than dilatation of the left ventricle was found.* In none of these cases was the snapping first sound, so common in mitral stenosis, recorded during life. This finding of presystolic murmurs in various conditions involving left ventricular hypertrophy (nephritis, arteriosclerosis) is entirely in accord with my own experience and tends to show that the Austin Flint murmur is due to the enlarged left ventricle characteristic of aortic regurgitation and not to the regurgitation itself.

All this resolves itself for me into the belief that *when the heart is much enlarged nothing that you hear at the apex is of much importance as evidence of valvular disease*, except the rare axillary diastolic murmur of aortic regurgitation. *Systolic and presystolic murmurs at the apex of a very large heart have little significance.*

It should be remembered that patients suffering from mitral stenosis are very frequently unaware of any cardiac trouble, and seek advice for anæmia, wasting, debility, gastric or pulmonary complaints. This is less often true in other forms of valvular disease. We should be especially on our guard in cases of supposed "nervous arrhythmia" or "tobacco heart," if there has been an attack of rheumatism or chorea previously. Such cases may present *no* complaints except the irregularity—yet may turn out to be mitral stenosis.

4. *Aortic Disease* (*Rheumatic Type*)

Rheumatic endocarditis usually occurs in early life, more commonly in women and most often attacks the mitral valve. The commonest cause of aortic disease on the other hand—syphilitic aortitis—occurs at all ages and attacks men much more often than women. Nevertheless rheumatic cases do occur at all ages and in both sexes, and do not always spare the aortic cusps by any means.

1. *Aortic Regurgitation*

When produced by rheumatic or septic endocarditis, the lesion which results in aortic regurgitation is always a *thickening*, *shortening* and *adhesion* of the cusps (see Fig. 146).

In the rheumatic type of aortic disease now to be described, mitral disease is usually present as well. Hence the signs of the aortic lesion may be much modified. I shall describe first the sign in cases of rheumatic aortic disease with relatively slight mitral trouble or none at all.

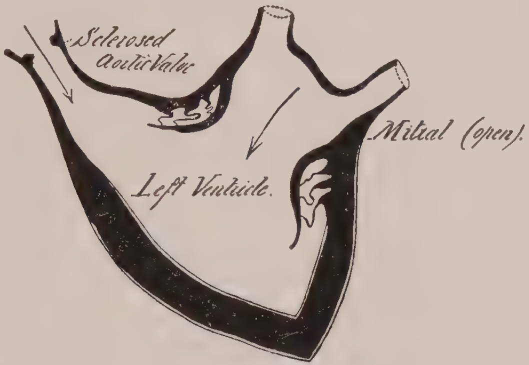

FIG. 146.—Diastole in Aortic Regurgitation. The blood is flowing back through the stumpy and incompetent aortic valves.

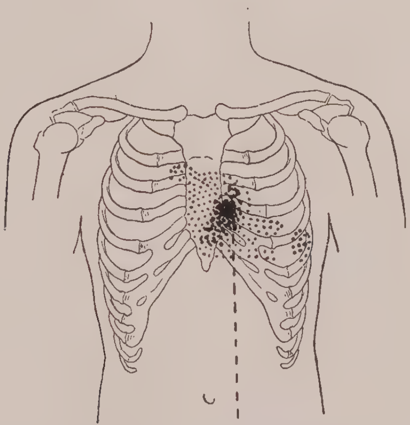

FIG. 147.—Position of the Point of Maximum Intensity of the Murmur of Aortic Regurgitation. The dots are most thickly congregated where the murmur is oftenest heard.

In rheumatic disease of the aortic valve, the arterial phenomena and the *cor bovinum* seen in syphilitic aortitis are less notable. The murmur is the most important evidence that we have. This murmur is sometimes difficult to hear. In the majority of cases, however, the characteristic *diastolic murmur* is easily heard if one listens in the right place, and when heard *it is the most distinctive and trustworthy of all cardiac murmurs.*

The murmur of aortic regurgitation, as has been already mentioned, is *diastolic* in time. *Its maximum intensity is usually not in the conventional aortic area* (*second right interspace*), *but on the left side of the sternum about the level of the fourth left costal cartilage.* In about one-fifth of the cases, and especially when the aortic arch is much dilated, the murmur is best heard in the conventional aortic area. Occasionally there are two points at which it may be loudly heard—one in the second right interspace and the other at or outside the cardiac apex, while between these points the murmur is faint. This is probably due to the fact that the left ventricle, through which the murmur is conducted, approaches the surface of the chest only at the apex, while the intermediate space is occupied by the right ventricle, which often fails readily to transmit murmurs produced at the aortic orifice. Less frequently the murmur of aortic regurgitation is heard with maximum intensity at the second or third left costal cartilage, at the apex, in the left axilla, or in the region of the ensiform cartilage.

From its seat of maximum intensity (*i.e.*, usually from the fourth left costal cartilage) the murmur is transmitted in all directions, but not often beyond the precordia. In about one-third of the cases it is transmitted to the left axilla or even to the back. It is sometimes to be heard in the subclavian artery and the great vessels of the neck; in other cases two heart sounds are audible in the carotid, but no murmur. The murmur is usually blowing and relatively high pitched, sometimes musical. Its intensity varies much, but is most marked at the beginning of the murmur, giving the impression of an *accent* there. It may occupy the whole of diastole or only a small portion of it—usually the earlier portion (see Fig. 148). Late diastolic murmurs are rare. The murmur may or may not replace the second sound of the heart. Broadbent believes that when it does not obliterate the second sound, the lesion is usually less severe than when only the murmur is to be heard. Allbutt dissents from this opinion.

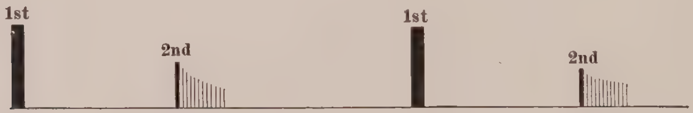

FIG. 148.—Short Diastolic Murmur Not Replacing the Second Sound.

The position of the patient's body has but little effect upon the murmur—less than upon murmurs produced at the mitral orifice.

A systolic murmur is almost always present as well—and best heard in the second right space. It may be due to the almost invariably accompanying *aortic stenosis* (see below).

Differential Diagnosis.—The chief point of difficulty is to distinguish the relatively rare (rheumatic) lesions of the aortic valve *alone* from those accompanying mitral stenosis. For in mitral stenosis we may have an early diastolic (not presystolic) murmur soft and blowing in quality along the left sternal border—in fact a precise imitation of the aortic regurgitant murmur (*Graham Steele murmur*). Differential diagnosis from an aortic lesion may then be impossible. True aortic disease (rheumatic type) is probable if we have in addition to the murmur a jerking or "Corrigan" type of pulse and the other arterial phenomena described in detail on page 250. But in rheumatic disease these arterial phenomena are often absent. We are then often unable to decide between mitral disease alone and mitral *plus* aortic disease, since the condition of the left ventricle does not help us but rather confuses us.

A loud diastolic murmur audible without a stethoscope 3 in. from the chest occurred in a recent case of "rheumatic" pericarditis with adhesions. Autopsy showed no valvular lesion whatever, only marked hypertrophy and dilatation.

2. *Aortic Stenosis*

Uncomplicated aortic stenosis, *i.e.*, without aortic regurgitation as well, is probably a myth. In the rheumatic type of heart disease the two lesions are almost always associated, though it may be impossible to find signs of regurgitation during life. It is probably cases of this latter type (unautopsied) that have given rise to the conception of a "pure" aortic stenosis. At any rate I shall consider it here wholly as an accompaniment of (rheumatic) aortic regurgitation. Out of two hundred and fifty-two autopsies made at the Massachusetts General Hospital in cases of valvular disease there was no one of uncomplicated arotic stenosis. Thirty cases occurred in combination with aortic regurgitation. Of these 19 or 63% were recognized in life and 11 or 36% were not recognized. *During life the diagnosis of aortic stenosis is frequently made, but often on insufficient evidence—i.e.,* upon the evidence of a systolic murmur heard with maximum intensity in the second right intercostal space and transmitted into the vessels of the neck. Such a murmur does indeed occur in aortic stenosis, but is by no means peculiar to this condition. Of the other diseases which produce a similar murmur more will be said under Differential Diagnosis.

For the diagnosis of aortic stenosis we need the following evidence:

(1) A systolic murmur heard best in the second right intercostal space.

(2) The characteristic pulse (*vide infra*).

(3) A palpable thrill (usually).

(4) Absence or great enfeeblement of the aortic second sound.[1]

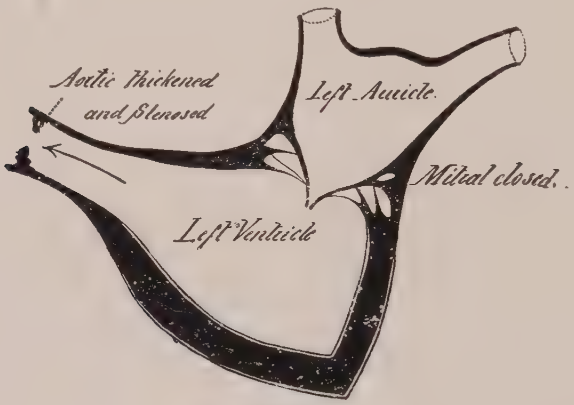

FIG. 149.—Aortic Stenosis. The heart is in systole and the blood column is obstructed by the narrowed aortic ring. The mitral is closed (as it should be).

Of these signs the thrill is the most important. The heart may or may not be demonstrably enlarged.

Each of these points will now be described more in detail.

(a) *The Murmur*

(*a*) The *maximum intensity* of the murmur, as has already been said, is usually in the second right intercostal space near the sternum or a little above that point near the sterno-clavicular articulation, but it is by no means uncommon to find it lower down, *i.e.*, in the third, fourth, or fifth right interspace, and occasionally it is best heard to the left of the sternum in the second or third intercostal space. (*b*) *The*

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[1] Against all reason I have twice seen at autopsy an aortic stenosis despite the fact that the "aortic second sound" had been loud in life.

time of the murmur is *late systolic;* that is, it follows the apex impulse at an appreciable interval, contrasting in this respect with the systolic murmurs heard loudest at the apex. (*c*) The murmur is usually *widely transmitted*, often being audible over the whole chest and occasionally over the skull and the arterial trunks of the extremities (see Fig. 150). It is usually heard less well over that portion of the precordia occupied by the right ventricle, while, on the other hand, it may be loudest in the region of the apex impulse, whither it is transmitted through the left ventricle. The same line of transmission was mentioned above as characteristic of the murmur of aortic regurgitation in many cases. The murmur is also to be heard over the carotids and subclavians, and can often be traced over the thoracic aorta along the spine and down the arms.

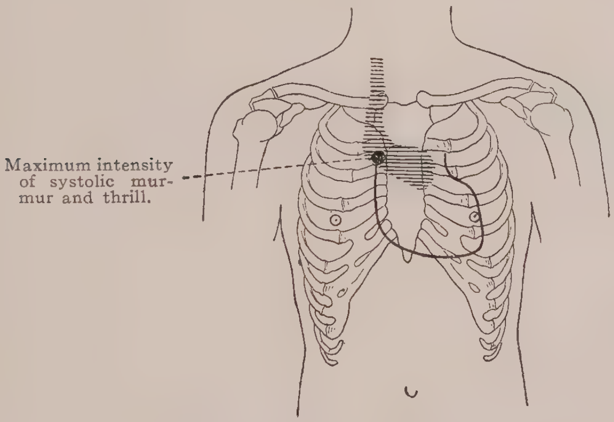

FIG. 150.—Aortic Stenosis. The murmur is audible over the shaded area and sometimes over the whole chest.

Until compensation fails the murmur is apt to be a very loud one, especially in the recumbent position; it is occasionally audible at some distance from the chest, and is often rough and vibrating, sometimes musical or croaking. Its length is usually great, extending throughout the whole of systole, but to this rule there are a good many exceptions.

(2) *The Aortic Second Sound*

The diminution or absence of the aortic second sound is an important point in the diagnosis of this lesion. It is true (as already mentioned) that we do not always get this sign. Yet without it diagnosis is always uncertain.

(*b*) *The Pulse*

Owing to the opposition encountered by the left ventricle in its attempt to force blood into the aorta, its contraction is apt to be prolonged; hence the pulse wave *rises* gradually and late, *and falls away slowly.* This is shown very well in sphygmographic tracings (see Fig. 151). But further, the blood thrown into the aorta by the left ventricle is prevented, by the narrowing of the aortic valves, from striking upon and expanding the arteries with its ordinary force; hence the pulse wave is not only slow to rise but *small in height*, sometimes contrasting with the powerful apex beat ("*pulsus parvus*") Again, the delay in the emptying of the left ventricle, brought about by the obstruction at the aortic valves, may render the contractions of the heart relatively *infrequent*, and hence the pulse is *infrequent* (*pulsus rarus*) as well as small and slow to rise. The "*pulsus rarus, parvus, tardus*" is therefore, an important point in diagnosis, but unfortunately it is to be felt in perfection only in the cases in which aortic stenosis is associated with very little regurgitation, *i.e.*, in the extreme cases with excessively narrow aortic orifice. When stenosis is combined with free regurgitation, as is usually the case, the above-described qualities of the pulse are modified as a result of the regurgitation. Indeed, I have in two cases observed a well-marked "Corrigan" pulse in life and been confronted *post-mortem* with a narrowed, rigid aortic valve!

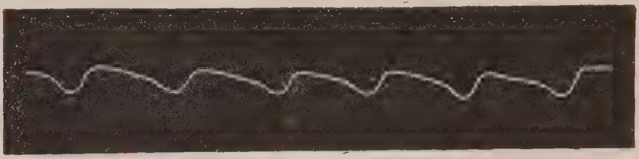

FIG. 151.—Sphymographic Tracing of the Pulse in Aortic Stenosis. Compare with the normal pulse wave and with that of aortic regurgitation (pages 251 and 252).

The slow, long pulse with a plateau at the summit is seen also in some cases of mitral stenosis and renal disease, and is not peculiar to aortic stenosis, but taken in connection with the other signs of the disease it has great value in diagnosis.

(*c*) *The Thrill*

In the majority of cases an intense purring systolic vibration may be felt if the hand is laid over the upper portion of the sternum, especially over the second right intercostal space. This thrill is continued into the carotids, can occasionally be felt at the apex, and rarely over a considerable area of the chest. It is a very important aid in the diagnosis of aortic stenosis, but is by no means pathognomonic, since aneurism may produce a precisely similar vibration of the chest wall.

The heart is slightly enlarged to the left and downward as a rule, but the apex impulse is unusually indistinct, "a well-defined and deliberate push of no great violence" (Broadbent) in some cases. In the majority it presents nothing peculiar or characteristic.

(*d*) *Diagnosis of Aortic Stenosis from the Etiology alone*

Post mortem experience has taught me that in practically all long standing cases of "rheumatic" endocarditis affecting the aortic valve, stenosis as well as regurgitation is present. Hence if these etiological factors can be recognized and if there is clear evidence of aortic regurgitaton, it is pretty safe to postulate the existence of aortic stenosis as well, *whatever the physical signs.*

(*e*) *Differential Diagnosis*

A systolic murmur heard loudest in the second right intercostal space is by no means peculiar to aortic stenosis, but may be due to any of the following conditions:

(*a*) Roughening, stiffness, fenestration, or slight congenital malformation of the aortic valves.

(*b*) Roughening or diffuse dilatation of the arch of the aorta.

(*c*) Aneurism of the aorta or innominate artery.

(*d*) Functional murmurs.

(*e*) Pulmonary stenosis.

(*f*) Open ductus arteriosus, and other congenital lesions.

(*a* and *b*) The great majority of systolic murmurs at the base of the heart, first appearing after middle life, are due to the causes mentioned above under *a*, *b*, and *c*. They are associated, as a rule, with high blood pressure and with evidence of arterio-sclerosis, chronic nephritis or syphilis and not with a "rheumatic" history. In such cases the murmur is usually combined with accentuation and ringing quality of the aortic second sound owing to the arterio-sclerosis and high arterial tension associated with the changes which produce the murmur. This *accentuation of the aortic second sound* enables us, except in rare cases, to exclude aortic stenosis, in which the intensity of the aortic second sound is almost always much reduced.

Diffuse dilatation of the aorta, which often accompanies arterio-sclerosis, is a frequent cause of a systolic murmur loudest in the second right interspace. This may be recognized in certain cases by the characteristic area of dulness on percussion or by x-ray.

Roughening of the intima of the aorta (*endaortitis*) is always be to suspected in elderly patients with calcified and tortuous peripheral arteries, and such a condition of the aorta doubtless favors the occurrence of a murmur, especially when accompanied by a slight degree of dilatation. The age, the presence of hypertension, the sharp aortic second sound, and the absence of a thrill and rheumatic history serve to distinguish such murmurs from those of aortic stenosis.

(*c*) Aneurism of the ascending arch of the aorta or of the innominate artery may give rise to every sign of aortic stenosis except the characteristic pulse and the diminution of the aortic second sound. In aneurism we may have a well-marked tactile thrill and a loud systolic murmur transmitted into the neck, but there is usually some abnormal pulsation to be felt, a characteristic x-ray shadow to be seen, and often some difference in the pulses or in the pupils, as well as a history of pain and symptoms of pressure upon the trachea and bronchi or recurrent laryngeal nerve. In aneurism the aortic second sound is usually loud, and the pulse shows none of the characteristics of aortic stenosis. Finally the evidence of syphilis and the absence of a "rheumatic" history may assist our decision.

(*d*) Functional murmurs, sometimes known as "hæmic," are occasionally best heard in the aortic area instead of in their usual situation (second left intercostal space). They are not accompanied by any palpable thrill, any diminution in the aortic second sound, or any distinctive abnormalities in the pulse.

(*e*) Pulmonary stenosis, a rare lesion, is manifested by a systolic murmur and by a thrill whose maximum intensity is usually on the left side of the sternum. In the rare cases in which this murmur is best heard in the aortic area it may be distinguished from the murmur of aortic stenosis by the fact that it is not transmitted into the vessels of the neck, has no effect upon the aortic second sound, and is not accompanied by the characteristic changes in the pulse. The electrocardiogram should show right sided predominance in pulmonary stenosis, left sided predominance in aortic stenosis.

(*f*) The murmur due to persistence of the ductus arteriosus may last through systole and into diastole; it may be accompanied by a thrill, but does not affect the pulse. Most of the other congenital lesions can be recognized by their history.

(*g*) The systolic murmur of aortic stenosis may be heard loudly at the apex, and hence the lesion may be mistaken for mitral disease.

But its association with a thrill and a long, slow pulse should enable us usually to differentiate the two lesions.

By the foregoing differentiæ aortic stenosis may be distinguished from the other conditions which resemble it, *provided it predominates over the regurgitation associated with it*, but unfortunately this is not very common. In many cases it is fairly well balanced or neutralized by the accompanying aortic regurgitation, and its characteristic signs are therefore obscured or greatly modified by the signs of the latter disease. In such cases the thrill and the "rheumatic" history are our chief point of support in the more or less reasonable suspicion that aortic stenosis is present.

Occasionally one can watch the development of an aortic stenosis out of what was formerly a pure regurgitant lesion, the stenosis gradually modifying the characteristics of the previous condition. One must be careful, however, to exclude a mitral stenosis which, as has been already mentioned above, is very apt to accompany cases of aortic disease, and which may modify the characteristic signs of aortic regurgitation very much as aortic stenosis does.

5. *Diseases of the Tricuspid and Pulmonary Valves*

1. *Tricuspid Regurgitation*

This lesion is in no way peculiar to the rheumatic type of heart disease. It occurs in all types as a phenomenon of broken compensation, but it is convenient to describe all its forms here along with the rarer rheumatic type.

Rheumatic endocarditis affecting the tricuspid valve is rare in post-fœtal life; in the fœtus it is not so uncommon. In cases of ulcerative or malignant endocarditis occurring in adult life, the tricuspid valve is occasionally involved, but the majority of cases of tricuspid disease occur in the following manner: Hypertrophy of the right ventricle occurs as a result of the mitral disease, and is followed in time by dilation; with this dilatation comes a stretching of the ring of insertion of the tricuspid valve, and hence a regurgitation through that valve. Tricuspid regurgitation, then, occurs in the latest stages of almost every case of mitral disease, and sometimes during the earlier attacks of failing compensation.

Out of 405 autopsies at Guy's Hospital in which evidence of tricuspid regurgitation was found, 271, or two-thirds, resulted from mitral disease, 68 from myocardial degeneration, 55 from pulmonary disease (bronchitis, emphysema, cirrhosis of the lung). Very few of these cases had been diagnosed during life, and in all of them the valve was itself healthy but insufficient to close the dilated orifice.

Gibson and some other writers believe that temporary tricuspid regurgitation is the commonest of all valve lesions, and results from weakening of the right ventricle in connection with states of anæmia, gastric atony, fever, and many other conditions. It is very difficult to prove or disprove such an assertion.

Tricuspid regurgitation is often referred to as serving like the opening of a "*safety valve*" to relieve a temporary pulmonary engorgement. This "safety-valve" action, however, may be most disastrous in its consequences to the organism as a whole, despite the temporary relief which it affords to the overfilled lungs. The engorgement is simply transferred to the liver and thence to the abdominal organs and the lower extremities, so that as a rule the advent of tricuspid regurgitation is recognized not as a relief but as a serious and probably fatal disaster.

(*a*) *Physical Signs*

(1) Systolic venous pulsation in the jugulars and in the liver.

(2) Engorgement of the right auricle producing an area of dulness beyond the right sternal margin.

(3) Intense cyanosis.

(4) Ascites and œdema of the legs.

(5) A systolic murmur heard loudest at or near the fifth left costal cartilage (a very uncertain and variable sign).

(1) Of chief importance in diagnosis is the presence of a systolic pulsation in the external jugular veins and of the liver, which unfortunately is not always present, but which when present is pathognomonic. I have already explained (see p. 89) the distinction between true *systolic* jugular pulsation and simple presystolic undulation or distention of the same veins, which has no necessary relation to this disease. The decisive test is the effort permanently to empty the vein by stroking it upward from below. If it instantly refills from below and continues to pulsate, tricuspid regurgitation is almost certainly present. If, on the other hand, it does not refill from below, the cause must be sought elsewhere.

Pulsation in the liver must be distinguished from the "jogging" motion which may be transmitted to it from the abdominal aorta or from the right ventricle. To eliminate these transmitted impulses one must be able to grasp the liver bimanually, one hand in front and one resting on the lower ribs behind, and to feel it distinctly expand with every systole, or else to take its edge in the hand and to feel it enlarge in one's grasp with every beat of the heart. Pressure upon the liver often causes increased distention and pulsation of the external jugulars if tricuspid regurgitation is present.

(2) Enlargement of the heart, both to the right and to the left, as well as downward, can usually be demonstrated. In rare cases a dilatation of the right auricle may be suggested by a percussion outline such as that shown in Fig. 152.

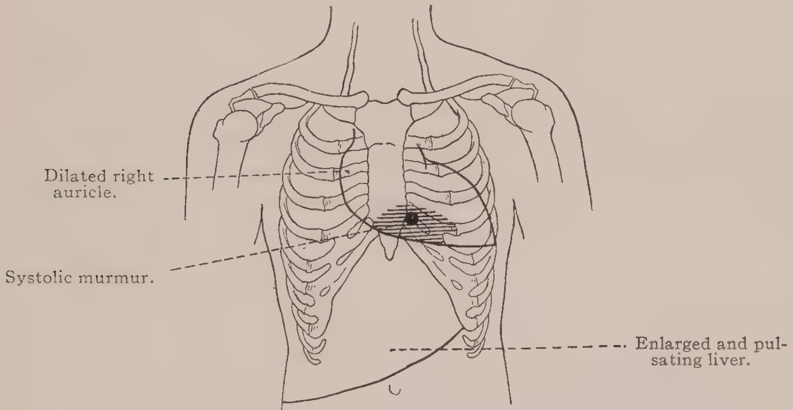

FIG. 152.—Tricuspid Regurgitation. The murmur is heard best over the shaded area.

The pulmonic second sound is usually not accented. The importance of this in differential diagnosis will be mentioned presently. If a progressive diminution in the intensity of the sound occurs under observation, the prognosis is very grave.

(3) Cyanosis is usually very great, and dyspnœa and general dropsy often make the patient's condition a desperate one.

(4) The murmur (systolic) is inconstant and usually indistinguishable from those produced at the aortic or mitral orifice.

(*b*) *Differential Diagnosis*

The cases autopsied at the Massachusetts General Hospital show that tricuspid regurgitation with stenosis is less often recognized during life than any other valvular lesion. The diagnosis was made ante mortem on only five out of twenty-nine cases. This is due to the following facts:

(*a*) Tricuspid regurgitation may be present and yet give rise to no physical signs which can be recognized during life.

(*b*) Tricuspid regurgitation occurs most frequently in connection with mitral disease; hence its signs are frequently masked by those of the latter lesion.

2. *Tricuspid Stenosis*

One of the rarest of "rheumatic" valve lesions is narrowing of the tricuspid valve. Only 28 cases (none recognized in life) have come under my observation, and in 1908, Herrick was able to collect but 187 cases from the world's literature. Out of 154 cases, 138, or 90 per cent., were combined with mitral stenosis, and only 12 times has tricuspid stenosis been known to occur alone.[1] These observations account for the fact that tricuspid stenosis has been recognized during life only 10 times since the murmur to which is gives rise is identical in time and quality, and nearly identical in position with that of mitral stenosis.

The heart is usually enlarged, especially in its transverse direction; the enlargement is greater than that of mitral stenosis, a fact which gives aid to our diagnosis.

The diagnosis is still further complicated in many cases by the presence of an aortic stenosis in addition to a similar lesion at the tricuspid and mitral valves, so that it seems likely that in the future, as in the past, the lesion will usually be discovered first at autopsy.

When we *know* we have marked mitral stenosis (perhaps with aortic stenosis as well) yet find *surprisingly little dyspnœa*, no accentuation of the pulmonic second sound, distended neck veins with anasarca and cyanosis, and when the mitral lesion does not improve as we should expect under treatment, and the heart is unusually large, we may be led occasionally to *suspect* tricuspid stenosis.

3. *Pulmonary Regurgitation*

Organic disease of the pulmonary valve is excessively rare in post-fœtal life, but may occur as part of an acute ulcerative or septic endocarditis. A *temporary* functional regurgitation through the pulmonary valve may be brought about by any cause producing very *high pressure in the pulmonary artery.* I have known two medical students with perfectly healthy hearts who were able, by prolonged holding of the breath, to produce a short, high-pitched diastolic murmur best heard in the second and third left intercostal spaces and ceasing as soon as the breath was let out. Of the occurrence of a murmur similarly produced under pathological conditions, especially in mitral stenosis, much has been written by Graham Steell.

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[1] Out of 87 cases collected from the post-mortem records of Guy's Hospital, 85, or 97 per cent., were associated with still more extensive mitral stenosis. At the Mass. General Hospital 26 of the 28 cases of tricuspid stenosis found at autopsy were associated with mitral stenosis as well.

From the diastolic murmur of aortic regurgitation we may distinguish the diastolic murmur of pulmonary incompetency by the fact that the latter is best heard over the pulmonary valve, is never transmitted to the apex of the heart nor to the great vessels, and is never associated with a Corrigan pulse nor with capillary pulsation. The right ventricle is hypertrophied, the pulmonic second sound is sharply accented and followed immediately by the murmur. Evidences of septic embolism of the lungs are frequently present and assist us in diagnosis. The regurgitation which may take place through the rigid cone of *congenital pulmonary stenosis* (see p. 274) is not recognizable during life.

6. *Combined Valvular Lesions*

It is essential that the student should understand from the first that the number of murmurs audible in the precordia is no gauge for the number of valve lesions. We may have four distinct murmurs, yet every valve sound except one. This is often the case in aortic disease—systolic and disastolic murmurs at the base of the heart, systolic and presystolic at the apex, yet no valve injured except the aortic. In such a case the systolic aortic murmur is due to roughening of the aortic valve. The systolic apex murmur results from relative mitral leakage (with a sound valve). The presystolic apex murmur is of the "Flint" type. Hence in this case *diastolic* murmur alone of the four audible murmurs is due to a valvular lesion.

It is a good rule not to multiply causes unnecessarily, and to explain as many signs as possible under a single hypothesis. In the above example the mitral leak might be due to an old endocarditis, and there *might* be mitral stenosis and aortic stenosis as well, but since we can explain all the signs as results—direct and indirect—of one lesion (aortic regurgitation) it is better to do so, and post-mortem experience shows that our diagnosis is more likely to be right when it is made according to this principle.

The most frequent combinations are:

(1) Mitral regurgitation with mitral stenosis.

(2) Aortic regurgitation with aortic stenosis, with or without mitral disease.

(3) Mitral and tricuspid stenosis.

1. *Double Mitral Disease*

(*a*) It very frequently happens that the mitral valve is found to be both narrowed and incompetent at autopsy when only one of these lesions had been diagnosed during life. In fact mitral stenosis is almost never found at autopsy without such a stiffening of the orifice as would produce an associated regurgitation, so that it is fairly safe to assume, whenever one makes the diagnosis of mitral stenosis, that mitral regurgitation is present as well, whether it is possible to hear any regurgitant murmur or not (see Fig. 153).

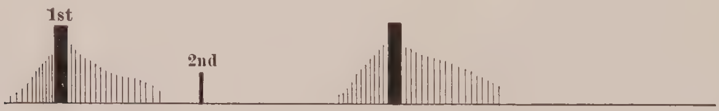

FIG. 153.—Mitral Stenosis and Regurgitation, showing relation of murmur to first heart sound.

(*b*) On the other hand, with a double mitral lesion one may have *only the systolic murmur* at the mitral valve and nothing to suggest stenosis unless it be a surprising sharpness of the first mitral sound. In chronic cases the changeableness of the murmurs, both in type and position, is extraordinary. One often finds at one visit evidences of mitral stenosis and at another evidences of a systolic mitral murmur alone. Either murmur may disappear altogether for a time and reappear subsequently. This is peculiarly true of the pre-systolic murmur, which is notoriously one of the most fleeting and uncertain of all physical signs.

As a rule the same inflammatory changes which produce a mitral systolic murmur in early life result, as they extend, in a narrowing of the mitral valve, so that the signs of stenosis come to predominate in later years. Coincidently with this narrowing of the diseased valve a certain amount of improvement in the patient's symptoms may take place, and Rosenbach regards the advent of stenosis in such a case as an attempt at regenerative or compensatory change. In many cases, however, no such amelioration of the symptoms follows.

2. *Aortic Regurgitation with Mitral Disease*

The signs of mitral disease occurring in combination with aortic regurgitation do not differ essentially from those of pure mitral disease except that the enlargement of the heart is apt to be more general and correspond less exclusively to the right ventricle (see Figs. 154 and 155.) The manifestations of the aortic lesion, on the other hand, are considerably modified by their association with the mitral disease. The Corrigan pulse is distinctly less sharp at the summit and rises and falls less abruptly. Capillary pulse is less likely to be present, and the throbbing of the peripheral arteries is less often visible.

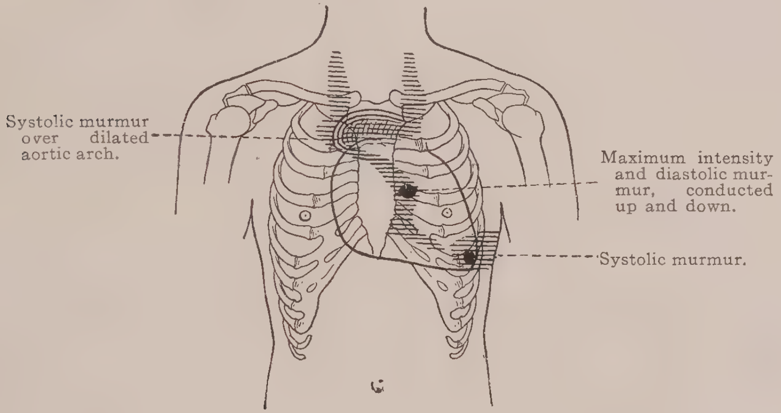
Systolic murmur over dilated aortic arch.
Maximum intensity and diastolic murmur, conducted up and down.
Systolic murmur.

FIG. 154.—Aortic and Mitral Disease. The shaded areas are those in which the murmurs are loudest.

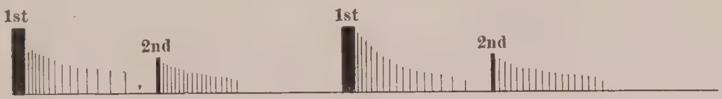

FIG. 155.—Showing Relation of Murmurs to Heart Sound in Disease of the Aortic and Mitral Valves.

3. *Aortic Regurgitation with Aortic Stenosis*

If the aortic valves are narrowed as well as incompetent, we find very much the same modification of the physical signs characteristic of aortic regurgitation as is produced by the advent of a mitral lesion; that is to say, the throbbing in the peripheral arteries is less violent, the characteristics of the radial pulse are less marked, and the capillary pulsation is not always to be obtained at all. Indeed, this blunting of all the typical manifestations of aortic regurgitation may give us material aid in the diagnosis of aortic stenosis, provided always that the mitral valve is still performing its function.[1]

(4) The association of mitral disease with tricuspid stenosis has been already described on p. 234.

PERICARDITIS

Three forms may be recognized clinically:

(1) Plastic, dry, or fibrinous pericarditis.
(2) Pericarditis with effusion (serous or purulent).
(3) Pericardial adhesions or adherent pericardium.

Fibrinous pericarditis may be fully developed without giving rise to any physical signs that can be appreciated during life. In several cases of pneumonia in which I suspected that pericarditis might be present, I have listened most carefully for evidences of the disease and been unable to discover any; yet at autopsy it was found fully developed—the typical shaggy heart. We have every reason to believe, therefore, that pericarditis is frequently present but unrecognized, especially in terminal streptococcus sepsis, in pneumonia and in the rheumatic attacks of children. On the other hand, it may give rise to very marked signs which are the result of—

(*a*) The rubbing of the roughened pericardial surfaces against one another when set in motion by the cardiac contractions.

(*b*) The presence of fluid in the pericardial sac.

(*c*) The interference with cardiac contractions brought about by obliteration of the pericardial sac together with the results of adhesions between the pericardium and the surrounding structures.

1. *Dry and Fibrinous Pericardium*

The diagnosis rests upon a single physical sign—"*pericardial friction*"—which is usually to be appreciated by auscultation alone, but may occasionally be felt as well. Characteristic pericardial friction is a rough, irregular, grating or shuffling sound which occurs irregularly and interruptedly during the larger part of each cardiac cycle. It is almost never accurately synchronous either with systole or diastole, but *overlaps* the cardiac sounds, and encroaches upon the pauses in the heart cycle. It is seldom exactly the same in any two successive cardiac cycles and differs thereby from sounds produced within the heart itself. Pericardial friction seems very near to the ear and may often be increased by pressure with the stethoscope; it is not materially influenced by the respiratory movements.

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[1] Some astonishing exceptions to this rule have been mentioned on pages 229 and 234.

It is best heard in the majority of cases in the position shown in Fig. 156; that is, over that portion of the heart which lies nearest to the chest wall and is not covered by the margins of the lungs; but not infrequently it may be heard at the base of the heart along the right sternal margin or over the whole precordial region. The sounds are fainter if the patient lies on the right side, and sometimes intensified if, while sitting or standing, he leans forward and toward the left, so as to bring the heart into closer apposition with the chest wall.

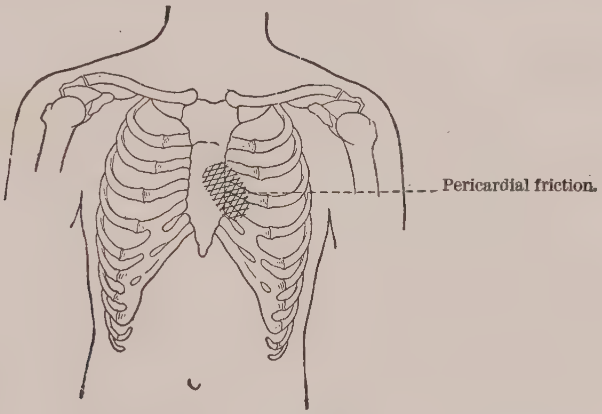

FIG. 156.—Showing Most Frequent Site of Audible Pericardial Friction.

Pericardial friction sounds often change rapidly from hour to hour, and may disappear and reappear in the course of a day.

In rare cases the friction may occur only during systole or only during diastole. In such cases the diagnosis between pericardial and intracardial sounds may be very difficult.

Differential Diagnosis

(*a*) *Pleuro-pericardial Friction*

Fibrinous inflammation affecting that part of the pleura which overlaps the heart may give rise to sounds altogether indistinguishable from those of true pericardial friction when the inflamed pleural surfaces are made to grate against one another by the movements of the heart. Such sounds are sometimes increased in intensity during forced respiration and disappear at the end of expiration, while true pericardial friction is usually best heard if the breath is held at the end of expiration. If a friction sound heard in the pericardial region ceases altogether when the breath is held, we may be sure that it is produced in the pleura and not in the pericardium, but in many cases the diagnosis cannot be made correctly.

(*b*) *Intracardiac Murmurs*

From murmurs due to valvular disease of the heart, pericardial friction can usually be distinguished by the fact that the sounds to which it gives rise do not accurately correspond either with systole or diastole, and do not occupy constantly any one portion of either of these periods. Cardiac murmurs are more regular, seem less superficial, and vary less with position and from hour to hour.

(*c*) *Friction Sounds of Unknown origin* (*dry surfaces?*)

In desiccated patients, *e.g.* in uræmia with vomiting, friction sounds like those of pericarditis may be heard yet no lesion is found *post-mortem.*

2. *Pericardial Effusion*

Following the fibrinous exudation, which roughens the pericardial surface and produces the friction sounds just described, serum may accumulate in the pericardial sac. Its quantity may exceed but slightly the amount of fluid normally present in the pericardium, or may be so great as to embarrass the cardiac movements and finally to arrest them altogether. In chronic (usually tuberculous) cases, the pericardium may become stretched so as to hold a quart or more without seriously interfering with the heart's action, while a much smaller quantity, if effused so rapidly that the pericardium has no time to accommodate itself by stretching, will prove rapidly fatal.

Hydropericardium denotes a dropsy of the pericardium occurring by transudation as part of a general dropsy in cases of renal disease or cardial weakness. The physical signs to which it gives rise do not differ from those of an inflammatory effusion, and, accordingly, all that is said of the latter in the following section may be taken as equally an account of the signs of hydropericardium.

Hæmopericardium, or blood in the pericardial sac, due to stabs or to ruptures of the heart, is usually so rapidly fatal that no physical signs are recognizable.

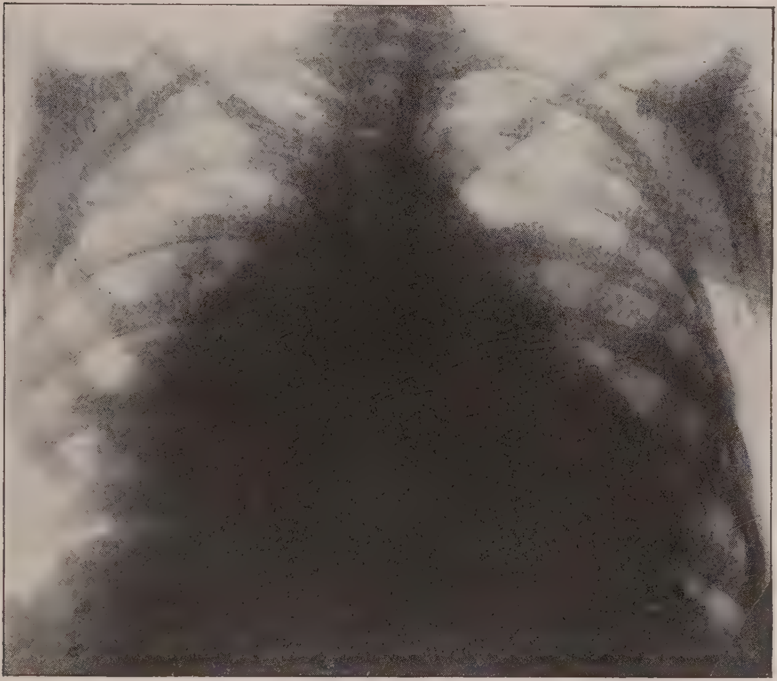

FIG. 157.—*X*-ray of Pericardial Effusion,

(*a*) *Physical Signs of Pericardial Effusion*

In most cases a pericardial friction rub has been observed prior to the time of the fluid accumulation. The presence of fluid in the pericardial sac is shown chiefly in four ways:

(1) By *percussion*, which demonstrates an area of dulness more or less characteristic (see below).

(2) By *auscultation*, which may reveal an unexpected feebleness in the heart sounds when compared with the power shown in the radial pulse.

(3) By the signs and symptoms of *pressure* exerted by the pericardial effusion upon surrounding structures.

(4) By the *x*-ray (see Fig. 157).

Bulging of the precordia is occasionally to be seen in children; in adults we sometimes observe a flattening of the interspaces just to the right of the sternum between the third and sixth ribs.

(1) *The Area of Percussion Dulness.*—The extent of the dull area depends not only on the size of the effusion and the position of the patient, but also on the amount of "give" in the pericardium and in the lungs, as well as on the size of the lingula pulmonalis. Allowing for these uncertain factors, we may say: (*a*) One of the most characteristic points is the unusual[1] extension of the percussion dulness a considerable distance to the left of the maximum cardiac impulse.

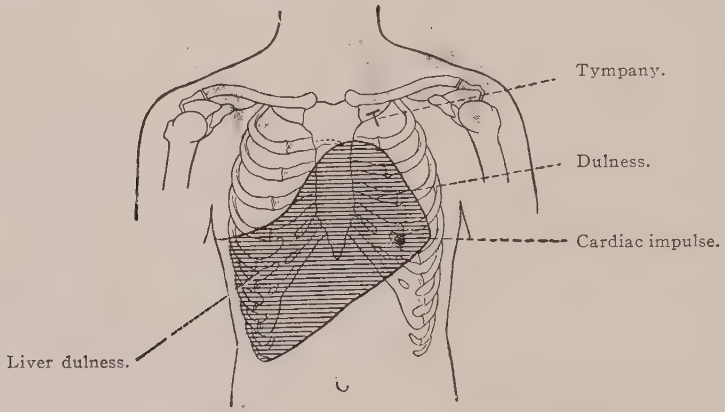

FIG. 158.—Percussion Dulness in Pericardial Effusion, with Tympanitic Resonance Under the Left Clavicle.

(*b*) The dullness to the right of the sternum may extend farther than in any type of cardiac hypertrophy and dilatation. It is only in pericardial effusion that we get so wide an area of dulness continuous with the heart (see Fig. 158). Rotch has called attention to the importance of dulness in the fifth right intercostal space as a sign of pericardial effusion, but a similar dulness may be produced by enlargement of the liver.

Except for the two points mentioned above (the unusual extension of the dulness to the left of the cardiac impulse, and the wide area to the right of the sternum), there seems to me to be nothing characteristic about the area of dulness produced by pericardial effusion. Even these signs are often unreliable. The sheet anchor for diagnosis in pericardial effusion is a good x-ray study.

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[1] In health the cardiac dulness extends about ¾ of an inch beyond the maximum cardiac impulse, but in pericardial effusion the difference is greater.

In some cases the area of dulness may be modified by change in the patient's position. After marking out the area of percussion dulness with the patient in the upright position, let him lie upon his right side. The right border of the area of dulness will sometimes move considerably farther to the right. A dilated heart can be made to shift in a similar way, but to a lesser extent. Comparatively little change takes place if the patient lies on his left side, and no important information is elicited by placing him flat on his back or by getting him to lean forward.

Unfortunately, it is only with moderate-sized effusions occurring in a pericardial sac free from adhesions to the surrounding parts that this shifting can be made out. Large effusions may not shift appreciably, and less than 150 c.c. of fluid probably cannot be recognized by this or by any other method. But with large effusions the lateral extension of the area of dulness may be so great as to be almost distinctive in itself, *i.e.*, from the middle of the left axilla nearly to the right nipple.

(2) Feebleness of the heart sounds and of the apex impulse is of diagnostic importance only when it gradually takes the place of the normal phenomena as one watches the heart from day to day. Under these conditions they have some confirmatory value in the diagnosis or pericardial effusion.

(3) Tubular breathing with dulness, increased voice sounds and tactile fremitus can often be heard near the angle of the left scapula. This is usually a result of lung compression, but a patch of pneumonia or a pleural effusion may produce almost identical signs.

A patch of tympanitic resonance is often to be found below the left clavicle, due no doubt to relaxation of the lung.

Pressure exerted by the pericardial exudation upon surrounding structures may also give rise to dyspnœa, especially of a paroxysmal type, to dysphagia, to aphonia, and to an irritating cough. The "paradoxical pulse," small and feeble during inspiration, is occasionally to be seen, but is by no means peculiar to this condition and has no considerable diagnosis importance. It is merely an exaggeration of pulse variations which often occur in health.

(4) *Inspection and palpation* usually helps us very little, but two points are occasionally demonstrable by these methods:

(*a*) A smoothing out of the intercostal depression in the precordial region, especially near the right border of the sternum between the third and the sixth ribs.

(*b*) A progressive diminution of the intensity of the apex impulse until it may be altogether lost. If this change occurs while the patient is under observation, and especially if the apex impulse *reappears* or becomes more distinct when the patient lies on the right side, it is of considerable diagnostic value. In conditions other than pericardial effusion, the apex impulse becomes *less* visible in the right-sided decubitus.

(*b*) *Differential Diagnosis*

(1) Our chief difficulty is to distinguish the disease from hypertrophy and dilatation of the heart. In the latter, which often complicates acute articular rheumatism with or without plastic pericarditis, the apex impulse is often very indistinct to sight and touch as in pericardial effusion. But the area of dulness is less likely to extend beyond the apex impulse to the left, or far beyond the right sternal edge, or to shift, when the patient lies on the right side. Pressure symptoms are less marked and there are usually no areas of broncho-vesicular breathing with tympanitic resonance under the left clavicle or in the back. *Yet not infrequently these differentiæ do not serve us, and the diagnosis can be made only by x-ray or by puncture.*

(2) I have twice known cases of encapsulated or interlobular empyema to be mistaken for pericardial effusion. In one case a needle introduced in the fifth left intercostal space below the nipple drew pus from what turned out later to be a localized purulent pleurisy, but the diagnosis was not made until a rib had been removed and the region thoroughly explored. It is not rare for pleuritic effusions to gather first in this situation, viz., just outside the apex impulse in the left axilla.

Such effusions may gravitate very slowly to the bottom of the pleural cavity or may become encapsulated and remain in their original and very deceptive position. In such cases the signs of compression of the left lung are similar to those produced by a pericardial effusion, and the results of punctures may be equivocal as in the case just mentioned. If there is *any* dulness, even a very narrow zone, in the left axilla between the fifth and eighth ribs, though there be none in the back, *x*-ray alone can set us straight.

As between pleuritic and pericardial effusion the presence of a good pulse and the absence of marked dyspnœa favors the former. In the two cases above referred to in which interlobar empyema was mistaken for pericarditis, the general condition of the patient struck me at the time as surprisingly good for pericarditis.

If *both* pleurisy and pericarditis are present, the area of pericardial dulness is not characteristic until the pleuritic fluid has been drawn off. The persistence of dulness in the cardio-hepatic angle and beyond the apex beat after a left pleurisy has been emptied by tapping, and after the heart has had time to return to its normal position, should make us suspect a pericardial effusion.

Despite the utmost care and thoroughness in physical examination, many cases of pericardial effusion go unrecognized, especially in infants, in elderly persons, or when the lung borders are adherent to the pericardium or to the chest wall.

In the rheumatic attacks of children, it should be remembered that *pericarditis is even more common than endocarditis.*

3. *Adherent Pericardium*

In the majority of cases the diagnosis cannot be made during life, unless the pericardium is adherent, not only to the heart, but to the walls of the chest as well. When this combination of pericarditis with chronic mediastinitis is present, the diagnosis may be suggested by:—

(*a*) A systolic retraction of the chest wall in the region of the apex impulse, at the base of the left axilla and in the region of the eleventh and twelfth ribs in the left back (Broadbent's sign). Such retraction is more marked during a deep inspiration. (It should be remembered that systolic retraction of the interspaces in the vicinity of the apex is very commonly seen in cases of cardiac hypertrophy from any cause, owing to the negative pressure produced within the chest by the contraction of a powerful heart.) A quick rebound of the cardiac apex at the time of diastole (the diastolic shock) is said to be characteristic of pericardial adhesions, but is often absent.

(*b*) Collapse of the cervical veins during diastole has been noticed by Friedreich, and the paradoxical pulse, above described, is said to be more marked in adherent pericardium than in any other known condition. Most recent writers, however, place no reliance upon it.

(*c*) Wm. H. Smith considers that the absence of any shift in the position of the apex beat wih respiration or change of patient's position, is an important point in favor of mediastino-pericarditis. In health and in valvular or parietal disease of the heart, the apex beat will swing from one to two inches to the left when the patient lies on his left side, and the descent of the diaphragm during full inspiration lowers the position of the cardiac impulse considerably.

(*d*) The presence of hypertrophy and dilatation affecting especially the left side of the heart, and not accounted for by the existence of any disease of the arteries, the cardiac valves, the lung, or of the kidney, should make us suspect pericardial and mediastinal adhesions.

In children adherent pericardium is perhaps the commonest cause of a *corbovinum* and in adults the largest hearts that I have seen have been due to the same cause.

(*e*) *X*-ray shows a restriction of the up-and-down movement of the heart during respiration. This is more marked and more characteristic than the limitation of lateral swing with change of position.

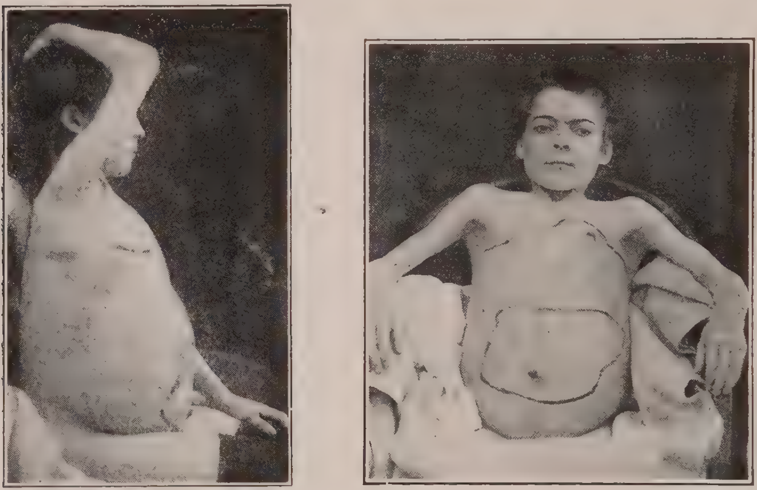

FIG. 159.—Adherent Pericardium, Ascites. D D, Dull area; R R, Resonant.

Inspection of the tissues just below the ensiform cartilage may show a complete arrest of the normally slight movements of this part of the abdominal wall.

(*f*) Adherent pericardium, occurring as a part of a widespread chain of fibrous processes involving the pleura, the mediastinum, and the peritoneum, may give rise in young persons to a train of symptoms and signs suggesting cirrhosis of the liver. Ascites collects, the liver is enlarged, yet there are no signs in the heart, kidneys, or blood sufficient to explain the condition. In any such case adherent pericardium should be considered. Fig. 159 shows the appearance in cases of this kind in which the diagnosis was verified by autopsy.

Summary

The diagnosis of adherent pericardium with chronic mediastinitis is suggested by

(*a*) Systolic retraction of the lower intercostal spaces in the left axilla and in the left back, followed by a diastolic rebound.

(*b*) The absence of any change in the position of the apex impulse with respiration or change of position.

(*c*) The presence of marked hypertrophy and dilatation of one or both ventricles without obvious cause.

(*d*) The absence of any respiratory excursion of the heart (*x*-ray) or of the abdominal wall at the costal angle.

(*e*) The presence of signs like those of hepatic cirrhosis in a young person and without any obvious cause.

CHAPTER XIII

SYPHILITIC HEART DISEASE

The border lines separating syphilitic infection of the cardio-vascular system from the other types of heart disease are not yet clearly defined. The painstaking researches of Warthin[1] have shown that spirochætes can be demonstrated in the heart of many cases of "myocarditis" and "arterio-sclerosis" especially if one studies minutely the tissues about the blood vessels. From Warthin's studies it appears wise to suspect that the heart is syphilitic in every patient showing good evidence of syphilis elsewhere. Cases of myocardial weakness or arterio-sclerosis with a positive Wassermann reaction may possibly be classed as syphilitic heart disease and doubtless a vague but considerable proportion of all the cases described in Chapter XV are in fact syphilitic.

Gumma of the heart wall has often been found post-mortem but can rarely be recognized in life. It may be suspected, when in a case of florid syphilis there arise sudden and marked evidences of cardiac weakness without obvious valve lesions or when extensive heart block occurs in a syphilitic.

The only type of syphilitic cardio-vascular disease now clearly recognizable in life is

I. SYPHILITIC AORTITIS WITH OR WITHOUT ANEURISM

Aortitis can be recognized only when it produces

(*a*) Aortic regurgitation.

(*b*) Aneurism.

In these forms it makes up about 12 per cent. of the hospital cases of heart disease. Each of these will now be described in detail.

1. *Aortic Regurgitation* (*Syphilitic Type*)

Seventy per cent. of my cases have occurred in men, the average age being 47. Whenever we find aortic regurgitation in a young or middle aged man with no rheumatic history, syphilis is to be suspected. This type of disease is especially common in negroes. The Wassermann reaction is of course a most important aid in diagnosis.

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[1] Transactions of the Assoc. of American Physicians, 1914, p. 416.

Since in syphilitic aortitis the regurgitation is due to a primary infection of the aortic arch extending down to the aortic valves and producing a dilatation of the aortic ring, there is rarely any other valve lesion to complicate the picture. The aortic valves rarely, if ever, fuse to produce a stenosis (as in "rheumatic" aortic disease), and there is no mitral stenosis to limit the amount of blood thrown into the aorta. Hence it is in syphilitic aortitis that we get the classical picture of aortic regurgitation, full blown and in its typical form. In rheumatic disease of the aortic valve the signs are more or less masked by other lesions.

2. *Physical Signs*

Inspection reveals more that is important in this disease than in any other valvular lesion. In extreme cases the patient's face or hand may blush visibly with every systole. Not infrequently one can make the diagnosis across the room or in the street by noting the violent throbbing of the carotids, which may be such as to shake the person's whole head and trunk, and even the bed on which he lies. No other lesion is so apt to cause a heaving of the whole chest and a bobbing of the head, and no other lesion so often causes a bulging of the precordia, for in no other lesion is the enlargement of the heart so great (*cor bovinum* or ox-heart). The throbbing of the dilated aorta can often be felt and sometimes seen in the suprasternal notch or in the second right interspace. Not only in the carotids but the subclavians, the brachials and radials, the femoral and anterior tibial, and even the digital and dorsalis pedis arteries may visibly pulsate, and the characteristic jerking quality of the pulse may be seen as well as felt. This visible pulsation in the peripheral arteries, while very characteristic of aortic regurgitation, is occasionally seen in cases of simple hypertrophy and dilatation of the heart from chronic nephritis as well as in arteriosclerosis, Graves' disease, and in severe anæmias. If the arteries are extensively calcified, their pulsation may become much less marked.

The peculiar conditions of the circulation whereby it is "changed into a series of discontinuous discharges as if from a catapult" (Allbutt) throws a great tensile strain upon all the arteries, and results, in almost every long-standing case, in increasing both their length and their diameter. The visible arterial trunks become tortuous and distended, while the arch of the aorta is diffusely dilated so as to resemble an aneurism (see Fig. 160). With each heart beat the snaky arteries are often jerked to one side as well as made to throb.

Inspection of the region of the cardiac impulse almost always shows a very marked displacement of the apex beat both downward and outward (but especially the former), corresponding to the hypertrophy and still more to the dilatation of the left ventricle, which is usually very great, and to the downward sagging of the enlarged aorta. Dilatation is in this disease an essentially helpful and compensatory process. Not at all infrequently one finds a systolic *retraction* of the interspaces near the apex beat instead of a systolic *impulse.* This is probably due to the negative pressure produced within the chest by the powerful contraction of an hypertrophied heart. In the suprasternal notch one often feels, as well as sees, a marked systolic pulsation transmitted from the arch of the dilated aorta, and sometimes mistaken for saccular aneurism.

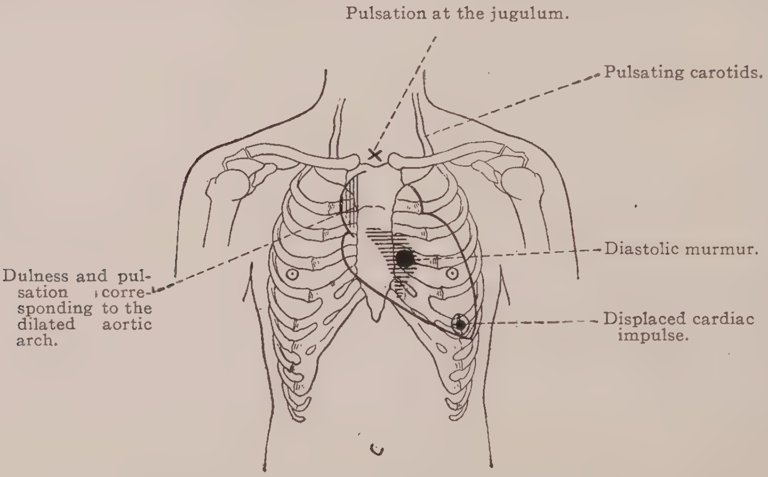
Pulsation at the jugulum.
Pulsating carotids.
Diastolic murmur.
Dulness and pulsation corresponding to the dilated aortic arch.
Displaced cardiac impulse.

FIG. 160.—Aortic Regurgitation, Showing Position of the Diastolic Murmur and Areas of Visible Pulsation.

3. *Capillary Pulsation*

If one passes the end of a pencil or other hard substance once or twice across the patient's forehead, and then watches the red mark so produced, one can often see a systolic flushing of the hyperæmic area with each beat of the heart. This is by far the best method of eliciting this phenomenon. It may also be seen if a glass slide is pressed against the mucous membrane of the lip so as partially to blanch it, or if one presses upon the finger-nail so as partially to drive the blood from under it; but in both these manœuvres error may result from inequality in the pressure made by the observer upon the glass slide or upon the nail. Very slight movements of the observer's fingers, even such as are caused by his own pulse, may give rise to changes simulating capillary pulsation. Capillary pulsation of normal tissues is not often seen in any condition other than aortic[1] regurgitation, yet occasionally one meets with it in diseases which produce very low tension of the pulse, such as Graves' Diesease, phthisis or typhoid, anæmic and neurasthenic conditions, and I have twice seen it in perfectly healthy persons. In such cases the pulsation is usually less marked than in aortic regurgitation.

4. *Palpation*

Palpation verifies the position of the cardiac impulse and the heaving of the whole chest wall suggested by inspection. The shock of the heart is very powerful and deliberate unless dilatation is extreme, when it becomes wavy and diffuse. In the supraclavicular notch a systolic thrill is often to be felt. A diastolic thrill in the precordia is very rare.

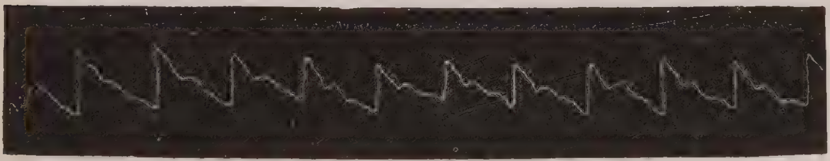

FIG. 161.—Sphygmographic Tracing from Normal Pulse.

The pulse is important, usually characteristic. The wave rises very suddenly and to an unusual height, then collapses completely and with great rapidity (pulsus celer) (see Figs. 161, 162).

This type of pulse, which is known as the "Corrigan pulse" or "water-hammer pulse," is exaggerated if one raises the patient's arm above the head so as to make the force of gravity aid in emptying the artery. The quality of the pulse in aortic regurgitation is due to the fact that a large volume of blood is suddenly and forcibly thrown into the aorta by the hypertrophied and dilated left ventricle, thus causing the characteristically sharp and sudden rise in the peripheral arteries. The arteries then empty themselves *in two directions at once*, forward into the capillaries and backward into the heart through the incompetent aortic valves; hence the sudden collapse in the pulse which, together with its sharp and sudden rise, are its important characteristics. The arteries are large and often elongated so as to be thrown into curves.

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[1] Jumping toothache and throbbing felon are common examples of capillary pulsation in inflamed area.

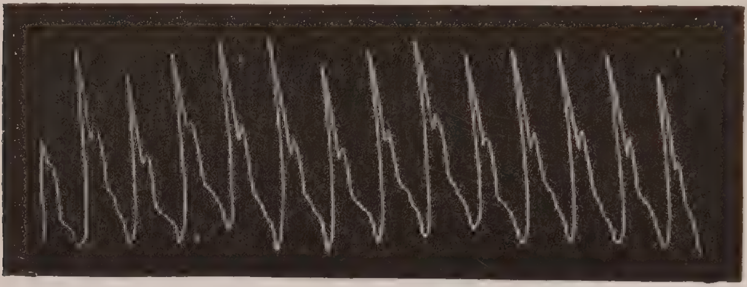

FIG. 162.—Sphygmographic Tracing of the "*Pulsus Celer*" in Aortic Regurgitation. Its collapsing character is well shown.

While compensation lasts, the pulse is usually regular in force and rhythm. *Irregularity is therefore an especially grave sign*, much more so than in any other valvular lesion.

Percussion

Percussion adds but little to the information obtained by inspection and palpation, but verifies the results of these methods of investigation respecting the increased size of the heart, and especially of the left ventricle, which may reach enormous dimensions, especially in cases occurring in young persons. The heart may be increased to *more than four times its normal weight.*

Auscultation

Unless the free ear is used, the diastolic murmur may be so faint as to be easily overlooked. This is especially true in cases occurring in elderly people, and when the patient has been for a considerable time at rest. The difficulty of recognizing certain cases of aortic regurgitation during life is shown by the fact that out of sixty-eight cases of aortic regurgitation demonstrated at autopsy in the Massachusetts General Hospital, only fifty-seven or 84 per cent. were recognized during life. Further description of the aortic regurgitant murmur of syphilitic disease is unnecessary since it is identical with that also described under rheumatic disease of the aortic valve on pages 223 and 224.

Over the large peripheral arteries, especially the femoral, one hears in most cases a sharp, short, systolic sound ("pistol-shot sound") due to the sudden filling and tautening of a relaxed artery. This sound is merely an exaggeration of what may often be heard in health.

Pressure with the stethoscope will also bring out a systolic murmur (as in health also) and occasionally a *diastolic* murmur as well (*Duroziez's sign*). The last murmur is practically never heard except in aortic regurgitation.

Summary and Differential Diagnosis

A *diastolic murmur* heard with the maximum intensity about the *fourth left costal cartilage* (less often in the second right interspace) gives us almost complete assurance of the existence of aortic regurgitation. From mitral stenosis and from pulmonary regurgitation, an exceedingly rare lesion, the disease is distinguished by the presence of predominating hypertrophy of the left ventricle with a heaving apex impulse and by the following *arterial phenomena:*

(*a*) Visible pulsation in the peripheral arteries.
(*b*) Capillary pulsation.
(*c*) "Corrigan" pulse.
(*d*) "Pistol-shot sound" in the femoral artery.
(*e*) Duroziez's sign.
(*f*) High pulse-pressure (see above, p. 114).

Cardiopulmonary murmurs (see page 192) are occasionally diastolic, but are very markedly influenced by position and by respiration, while aortic murmurs are but slightly modified.

The very rare functional diastolic murmur, transmitted from the veins of the neck and heard over the base of the heart in cases of grave anæmia, may be obliterated by pressure over the bulbus jugularis. Such pressure has no effect upon the murmur of aortic regurgitation. I have reported (*Johns Hopkins Bull.*, May, 1903) three cases of intense anæmia associated with diastolic murmurs exactly like those of aortic regurgitation, but proved post mortem to be independent of any valvular lesion. The arterial phenomena were not marked, but the diagnosis of such cases is very hard. Luckily they are rare. The origin is obscure. It must be remembered that aortic regurgitant murmurs are often exceedingly faint, and should be listened for with the greatest care and under the most favorable conditions.

(c) *Estimation of the Extent and Gravity of the Lesion*

The extent of the lesion is roughly proportional to—

(*a*) The amount of hypertrophy of the left ventricle.

(*b*) The degree to which the pulse collapses during diastole (provided the radial is not so much calcified as to make collapse impossible).

(*c*) The degree to which the murmur replaces the second sound as heard over the right carotid artery (Broadbent).

Irregularity of the pulse is a far more serious sign in this disease than in lesions of the mitral valve, and indicates the beginning of a serious failure of compensation.

Another grave sign is a diminution in the intensity of the murmur.

II. COMPLICATIONS

(1) *Aneurism* (see p. 256).—Aneurismal dilatation of the aortic arch is usually associated with aortic regurgitation and may produce a characteristic area of dulness to the right of the sternum (see Fig. 160). Not infrequently this dilatation is the cause of a systolic murmur to be heard over the region of the aortic arch and in the great vessels of the neck.

(2) *Roughening of the Aortic Valves.*—In almost all cases of aortic regurgitation the valves are sufficiently roughened to produce a systolic murmur as the blood flows over them. This murmur is heard at or near the conventional aortic area, and may be transmitted into the carotids. (The relation of these murmurs to the diagnosis of aortic stenosis has been considered with the latter lesion.)

(3) The return of arterial blood through the aortic valves into the left ventricle produces in time both hypertrophy and dilatation of this chamber, and results ultimately in a stretching of the mitral orifice which renders the mitral curtains incompetent. The result is a "*relative mitral insufficiency*," *i.e.*, one in which the mitral valve is intact but too short to reach across the orifice which it is intended to close. Such an insufficiency of the mitral occurs in most well-marked cases, it temporarily relieves the overdistention of the left ventricle

and often the accompanying angina, although at the cost of engorging the lungs.[1]

(4) *The Austin Flint Murmur.*—The majority of cases of aortic regurgitation are accompanied by a presystolic murmur at the apex. (For a fuller discussion of this murmur see above, p. 220).

(5) *Aortic stenosis* frequently accompanies cases of aortic regurgitation, especially in the rheumatic, choreic and tonsillar types occurring in young persons. It has the effect of increasing the intensity of the diastolic murmur, since the regurgitating stream has to pass through a smaller opening. Whether syphilitic aortitis can also produce aortic stenosis I am not certain. So far I have not seen a case proved post-mortem.

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[1] This relative insufficiency of the mitral valve has been termed its "safety-valve" action, but the safety is but temporary and dearly bought.

CHAPTER XIV

SYPHILITIC AORTITIS WITH ANEURISM

For clinical purposes thoracic aneurisms may be divided into the *diffuse* and the *saccular*. Saccular aneurisms of the ascending or descending portion of the arch of the aorta are apt to penetrate the chest wall, while aneurism of the transverse aorta or diffuse dilatations of the whole aortic arch are more likely to extend within the chest without eroding the thoracic bones. Practically any aneurism which penetrates the thoracic bones may be inferred to be saccular, but if no such penetration takes place, it may be impossible to make out whether the dilatation is diffuse or circumscribed. I shall consider:

I. The signs of the presence of aneurism.
II. The evidences of its seat.

I. Inspection and Palpation

1. *Abnormal Pulsation*

Inspection and *palpation* give us most of the important information in the diagnosis of aneurism. The patient should be placed in the position shown in Fig. 163, so that the light will strike obliquely across the surface of the chest, and the observer should be so placed that his eyes are as nearly as possible at the level of that part of the chest at which he expects to see pulsation.

In the majority of cases of aneurism some *abnormal pulsation* may be made out either to the right of the sternum in front, or in the region of the left scapula behind. If the aneurism is large, a considerable area of the chest wall may be lifted with each beat of the heart; with smaller growths the pulsating area may be small and sharply circumscribed. Not infrequently an abnormal pulsation at the sternal notch or in the neck may also be observed. Other causes of abnormal pulsations in the chest, such as dislocation or uncovering of the heart, must of course be excluded.

Palpation controls the results of inspection, but at times a pulsation may be seen better than felt; at others may be felt better than seen.

2. *Tumor*

If the aneurism involves the ascending portion of the aortic arch, it is likely sooner or later to erode the right margin of the sternum, and adjacent parts of the second or third costal cartilages, and appear externally as a round swelling in which a systolic pulsation is to be seen and felt. This pulsation is in some cases distinctly *expănsile* in character, and differs in this respect from the up-and-down motion which may be communicated to a tumor of the chest wall by the beating of a normal aorta. The tumor is usually firm, rarely soft, and may be as hard as any variety of malignant new growth. Occasionally the thickness of the lamellated clot within it is so great that no pulsations are transmitted to the surface.

3. *Thrill*

Whether the aneurism penetrates the chest or not, it is often possible to feel over it a *vibration thrill, systolic* in time. If the layer of lamellated clot in the sac is very thick, the thrill is less apt to be felt.

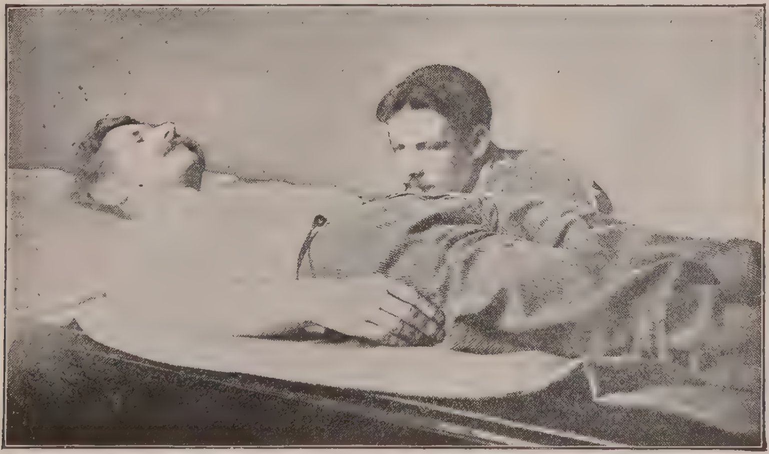


FIG. 163.—Position When Looking for Slight Aneurismal Pulsation.

4. *Diastolic Shock*

Of minor value in diagnosis is a *diastolic tap* which is appreciated by laying the palm of the hand lightly over the affected area. This diastolic shock is due to the recoil of the blood in the dilated aorta, and is felt in a minority of cases. (It also occurs in hypertension from any cause.) As the wall of the sac becomes weaker, the intensity of the shock diminishes. It may be appreciated over the trachea also, and is thought by some to have even more significance when felt in this situation.

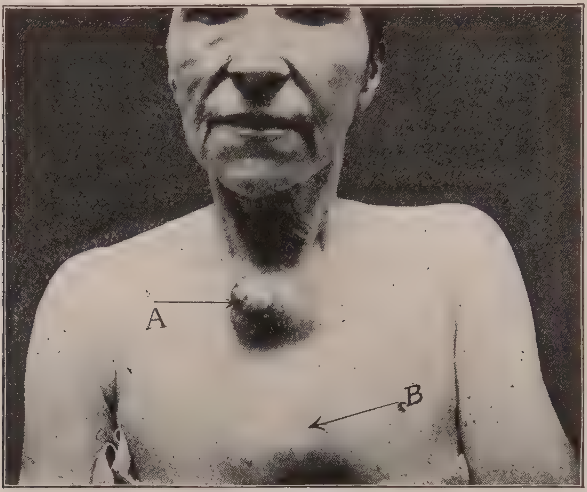


FIG. 164.—Aneurismal Tumor (*A*). The arrow *B* points to a gummatous swelling near the ensiform cartilage. The radiographic appearances of this case are shown below (Fig. 167).

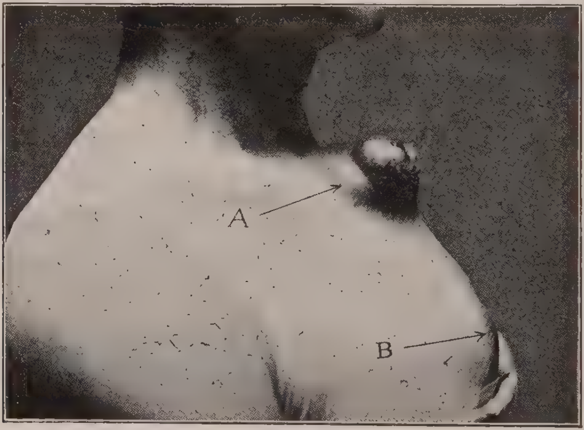


FIG. 165.—Aneurism Tumor Perforating the Sternum at *A*. At *B* there is a gummatous mass. (See below, Fig. 167, a radiograph of this case).

Of special importance in aneurism of the transverse arch is the sign known as the *tracheal tug*. The arch of the aorta runs over the left primary bronchus in such a way that when the aorta is dilated, the bronchus is pressed upon with each expansile pulsation of the artery.

5. *Tracheal Tug*

This systolic pressure transmitted to the trachea produces a distinct downward tug upon it with each systole of the heart. The tug is best felt by making the patient throw back his head so as to put the trachea upon a stretch. The physician then stands behind him and gently presses the tips of the fingers of both hands up under the lower border of the cricoid cartilage. In feeling thus for the tracheal tug as transmitted to the cricoid cartilage certain precautions must be observed:

(*a*) One must distinguish the tracheal tug from a simple pulsation transmitted to the superficial tissues by the vessels underneath. Such pulsation makes the tissues move *out and in* rather than up and down.

(*b*) A tracheal tug felt only during inspiration has no pathological significance and is frequently present in health.

While preparing to try for the tracheal tug we may notice whether there is any dislocation of the trachea, as shown by the displacement of Adam's apple. Stridor, cough, dysphagia, and other symptoms are produced by pressure on gullet and windpipe. Other signs of aneurism, due to the pressure of the dilated aorta upon the nerves or vessels of the mediastinum, are:

(1) Hoarseness or aphonia from pressure on the left recurrent laryngeal nerve.

(2) Inequality of the pupils.

(3) Inequality of the radial pulses.

(4) Œdema and cyanosis of one arm or of one side of the neck and head.

(5) Pain in one arm from the pressure of an aneurism involving the subclavian artery upon the brachial plexus.

(6) Evidence of pressure on the lung or on a bronchus.

Of these pressure signs the commonest and most important are: hoarseness, pain, and compression of the lung or bronchus.

Contraction or dilatation of the pupil is due to a paralytic or irritative affection of the sympathetic nerves. This symptom is much commoner than the other effect of pressure upon the sympathetic nerves; namely, flushing or sweating of one side of the face.

In comparing the pulses in the two radials we must bear in mind the possibility of a congenital difference between them, due to a difference either in the size of the arteries or in their position, and also that a tumor pressing on the subclavian may affect the pulse exactly as an aneurism. The pulse wave upon the affected side (most often the left) may be either less in volume or later in time than the wave in the other radial artery, according as the pulse wave is actually delayed in the aneurismal sac or merely diminished by it. In marked cases the pulse upon the affected side may be nearly or quite absent.

Examination of the heart itself may show some dislocation of the organ to the left and downward, owing to the direct pressure of the aneurismal sac, but no enlargement.

II. Percussion

If the aneurism is deep-seated, the results of percussion are negative. If, on the other hand, it be situated immediately beneath the sternum or close under the thoracic wall, an area of dulness, not present in the normal chest, may be mapped out. The outlines most commonly seen in such cases are shown in Fig. 166. When the aneurism involves the descending aorta, an area of dulness may be found in the region of the left scapula or below it, and pulsation may be detected in the same area.

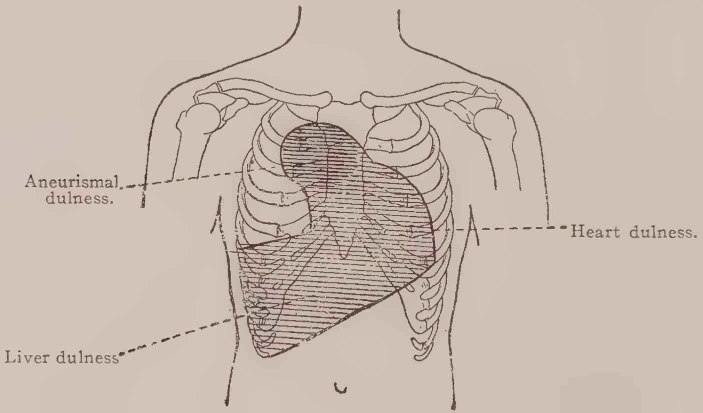


Fig. 168.—Diagram of Percussion Dulness in Aortic Aneurism.

III. Auscultation

The signs revealed by auscultation are not of much diagnostic value as a rule. In about one-half of the cases of sacculated aneurism there are no sounds or murmurs to be heard over the tumor. In other cases a systolic murmur, the audible counterpart of the vibratile thrill, may be heard over the area of pulsation, tumor, or dulness corresponding to the aneurismal sac. This systolic murmur may be due to many causes other than aneurism, and has nothing characteristic about it. A similar systolic sound is sometimes heard over the trachea (Drummond's sign) or in the mouth, if the patient closes his lips around the pectoral extremity of the stethoscope (Sansom's sign).

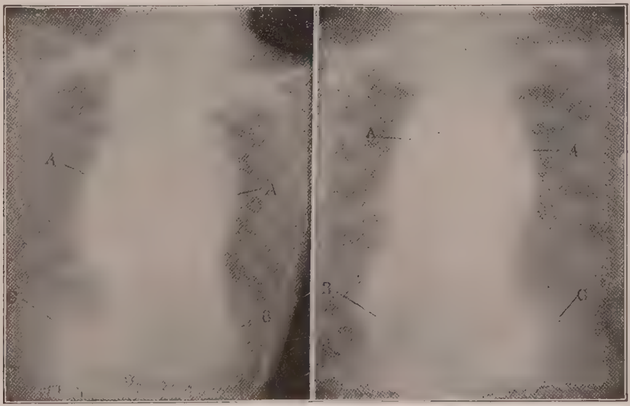
A loud diastolic murmur is audible in most cases as a result of an accompanying aortic regurgitation (see above).

If a portion of either lung is directly pressed upon by the aneurismal sac, we may have the signs of condensation of the lung in the area pressed upon (slight dulness, broncho-vesicular breathing, and exaggerated voice sounds). If one of the primary bronchi is pressed upon as occasionally happens, atelectasis of the corresponding lung may be manifested by the usual signs (dulness, absence of tactile fremitus and of respiratory and vocal sounds).

If the aneurismal sac is of very great size, the pulse wave in the femorals may be obliterated, as happened in a case described by Osler.

IV. Radioscopy

With the fluoroscope and through radiography one can often make out a shadow corresponding to the position of the aneurism.



From the front. From behind.

Fig. 167.—Radiograph of Case whose Photograph is Reproduced as Figs. 164 and 165. In the right-hand cut are shown the appearances seen from behind. The left hand cut, *A*, *A*, aneurismal sac; *B*, heart displaced; *C*, liver (not in focus).

Not infrequently this is the only convincing sign of the disease. Hence *x*-ray examination is of the utmost value. The position of the shadow is best explained by reference to Figs. 167, 168, 169, and 170.

Summary

The most important signs of aneurism are:

1. *Abnormal pulsation*—visible or palpable.
2. *Tumor* over which (3) a *thrill* can be felt.
4. *Tracheal tug.*
5. *Pressure signs* (unequal pulses, pupils, hoarseness, pain, etc.).
6. *Dulness* on percussion over the suspected area.

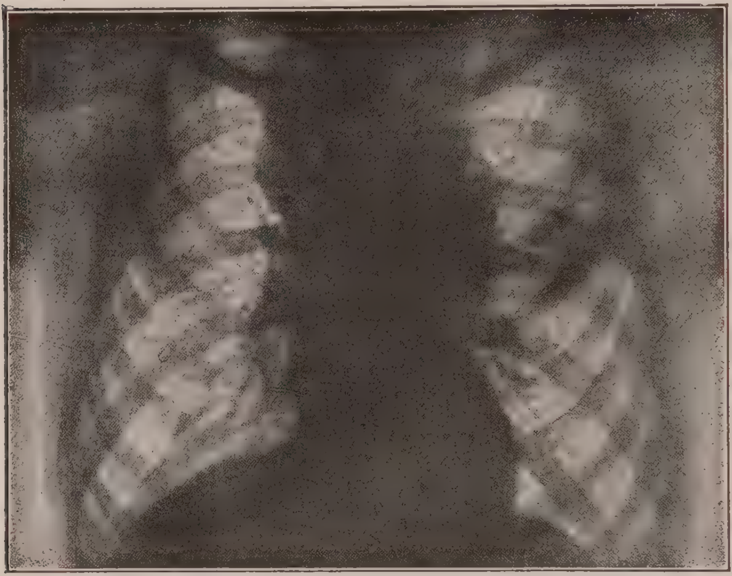


FIG. 168.—Aortic Aneurism. (From v. Ziemssen's Atlas.)

7. *Diastolic murmur* (aortic regurgitation).
8. *Systolic murmur* (least important of all).
9. *Radioscopy* may demonstrate a shadow higher up than that corresponding to the heart and extending beyond that produced by the sternum, spinal column, and great vessels.

V. DIAGNOSIS

1. *Diagnosis of the Seat of the Lesion*

(*a*) *Aneurism of the ascending arch* generally approaches or penetrates the chest wall in the vicinity of the *second right intercostal space* near the sternum. Previous to perforating the thoracic parietes the growth of the aneurism may give rise to pain, pulsation, and dulness and thrill in this region.

(*b*) *Aneurism of the transverse arch* or diffuse dilatation of the aorta, which is the most common of all types of aortic aneurism, may not give rise to any visible pulsation of the chest wall, and, if deep-seated, need not produce any abnormal dulness on percussion. In such cases an aneurism is to be recognized, if at all, by *x-ray or by evidences of pressure* on the nerves or vessels of the mediastinum (cough, aphonia, inequality of the pupils, tracheal tug, etc.).

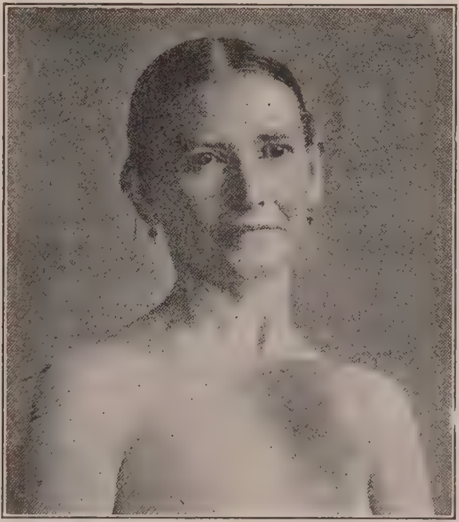


FIG. 169.—Aneurism of the Aorta. (Curschman.)

(*c*) *Aneurism of the descending aorta* gives rise, usually, to severe and persistent pain in the back, which radiates along the intercostal nerves or downward. Other pressure symptoms are not marked, but in advanced cases an area of abnormal dulness and pulsation may be found in the region of the left scapula or below it.

(*d*) If the *innominate artery* or one of the carotids is involved, we usually find a pulsating lump in the region of one or the other claviculo-sternal joint or at the root of the neck, and the trachea may be displaced to one side. This form, however, is distinctly rare. The violent throbbing and dilated carotid of aortic leakage is often mistaken for it.

2. *Differential Diagnosis*

(*a*) It is important to distinguish the diffuse dilatation of the aortic arch (not aneurism), which is present in most cases of hypertension with nephritis or arterio-sclerosis, from syphilitic aneurism of the transverse aorta. Dulness and even slight pulsation, perhaps with systolic murmur in the second and third right interspaces near the sternum, occur in many cases of chronic hypertension, but though the aorta is dilated, its coats are not ruptured and it never breaks. The *absence of pressure signs*, the negative Wassermann and the shape of the *x*-ray shadow distinguish it from syphilitic aneurism.

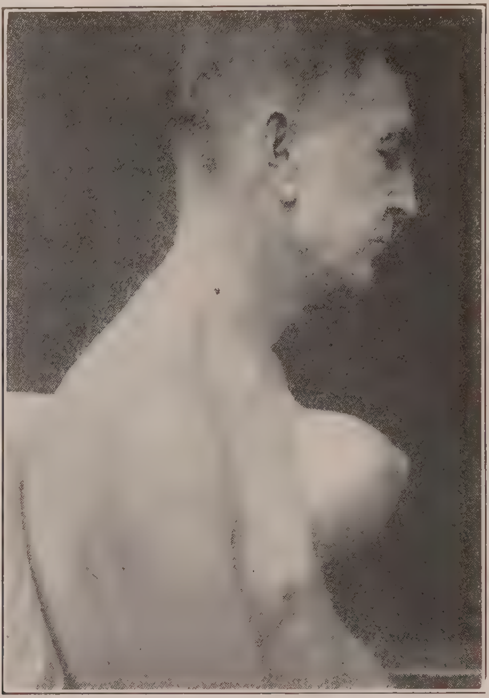


Fig. 170.—Aneurism of the Aorta.

(*b*) Aneurism is not infrequently mistaken for *aortic stenosis*, in which a systolic murmur and thrill, similar to those occurring in aneurism, are to be heard over the region of the aortic arch. From aortic stenosis aneurism is distinguished by the fact that it does not diminish the aortic second sound or produce characteristic changes in the pulse, and by the presence of some one of the symptoms above described, such as tracheal tug, pressure symptoms, etc. The "rheumatic" etiology of aortic stenosis, the *x*-ray, and the syphilitic basis of aneurism may often be recognized and help our diagnosis.

(*c*) Simple dynamic throbbing of a normal aortic arch similar to that which occurs in the abdominal aorta may lift the chest wall so as to simulate aneurism. The other positive symptoms and signs of aneurism are, however, absent.

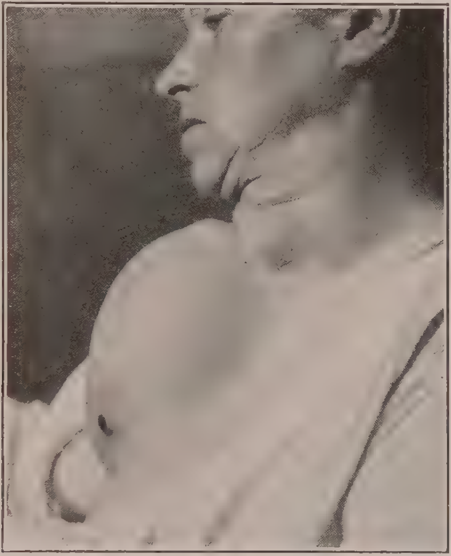


FIG. 171.—Aneurism of the Aorta with gumma.

(*d*) Pulmonary tuberculosis or cancer of the œsophagus, producing, as they may, substernal pain, cough, and aphonia by pressure upon mediastinal structures, have been mistaken for aneurism, from which, however, they may be distinguished by the absence of the positive signs above described, by the more rapid emaciation of the patient, and by the positive evidences of cancer or tuberculosis.

(*e*) *Empyema necessitatis* may produce a pulsating tumor like that of aneurism and the area of dulness may be similar, but there is no diastolic shock, no tactile thrill or murmur, and the history of the case and the *x*-ray shadow are usually very different from that of aneurism.

(*f*) *Mediastinal tumors* are sometimes mistaken for aneurism during life. They may produce a more intense and widespread dulness which is usually in the median line, while the dulness of aneurism is oftener at one side. The pulsation transmitted to a tumor by the heart has not the expansile character of aneurismal pulsation. Tumors are not associated with any diastolic shock, rarely with a tracheal tug. *X*-ray usually decides.

The course of most mediastinal tumors is progressive and attended by great cachexia, while the symptoms of aneurism are often more or less intermittent, and unless pain is severe there is no such emaciation or anæmia as is commonly seen with mediastinal tumors. Pressure symptoms may be the same in both diseases, but are usually more marked with mediastinal growths. A metastatic nodule over the clavicle sometimes betrays the presence of a primary focus within the chest.

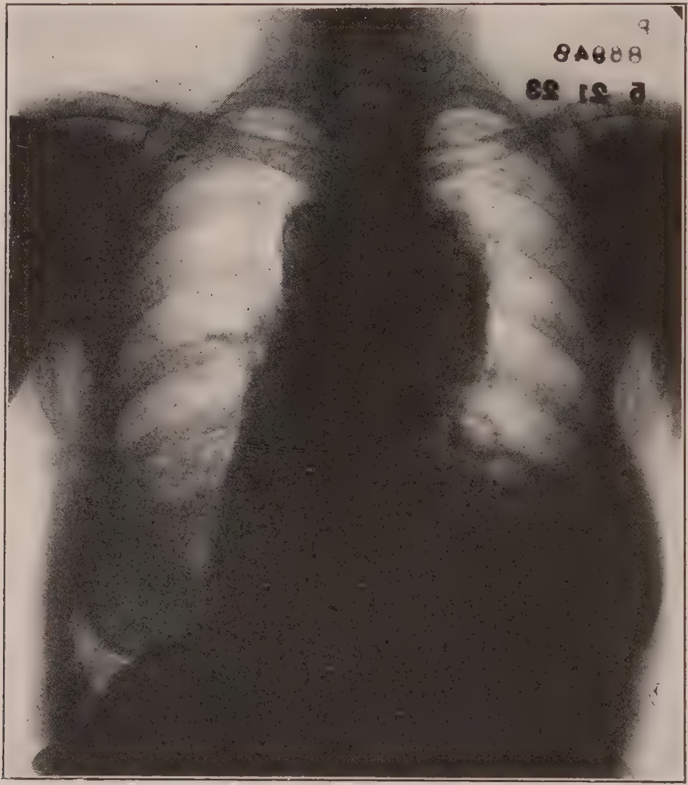


Fig. 172.—Neoplasm of mediastinum. Fluid at right base.

The most helpful differential points are the *x*-ray, the evidence of the presence or absence of syphilis, and of a diastolic murmur such as is usually present with aneurism and absent with neoplasm.

X-ray *sometimes* shows a definite expansile pulsation in the shadow corresponding to the sac. But I have found this piece of evidence as often wrong as right.

(*g*) *Retraction of the right lung* (fibroid phthisis), with or without displacement of the heart toward the diseased side, may uncover the heart so as to produce some of the signs of aneurism, *i.e.*, pulsation and dulness in the upper right intercostal spaces near the sternum, with a loud second sound and sometimes a systolic murmur in the dull area.

The history of the case and a careful examination of the lungs usually suffice to set us right.

(*h*) *Dilatation of the heart* may be so extreme that pulsation and percussion dulness appear in the characteristic aneurismal area to the right of the sternum, especially if there is solidification of the left lung. But the pulse is in such cases much weaker and more irregular than it is to be expected in uncomplicated cases of aortic aneurism, and the history of the case is usually decisive.

By the same marks we can distinguish the pulsations of a dilated heart, which sometimes appear in the left hypochondrium.

CHAPTER XV

HYPERTENSIVE CARDIO-VASCULAR DISEASE

Whether or not extra-renal arterio-sclerosis causes hypertension with cardiac hypertrophy and dilatation, or is the result of a primary hypertension (Allbutt's "Hyperpiesia"), there is a frequent association between them. Indeed, there is no commoner type of cardiac enlargement than that associated with arterio-sclerosis and hypertension. Hence, the term "hypertensive cardio-vascular disease" is often used as synonymous with the lesions which I am about to describe. "Chronic myocarditis" or "fibrous myocarditis" and "fatty heart" are poor terms for clinical use, since we know no clinical picture corresponding to them. One recognizes usually in this order:

1. Hypertension.
2. Enlarged heart (see below).
3. Disturbances of rhythm, of impulse production or impulse conduction.
4. Sclerosed arteries (especially the brachial).
5. Dyspnœa, muscular weakness, chronic cough (often miscalled "chronic bronchitis,"), and a gradual accumulation of fluid in the legs, possibly with angina pectoris.

I. Physical Signs

Beside the hypertension and the sclerosed arteries (which need no further description here) we have evidence of *cardiac hypertrophy and dilatation*. The apex impulse is displaced downward and to the left. Its movement is slow and deliberate ("heaving impulse") so long as compensation remains fair. The aortic second sound is usually loud and ringing even in the cases when the systolic blood-pressure is not much elevated.

Diffuse dilatation of the aortic arch can often be made out by the *x*-ray, sometimes by percussion. The *x*-ray shadow and the aortic dullness are often 8 to 10 cm. wide.

Systolic murmurs are almost invariably audible at the base of the heart and at the apex. Whether one or two systolics are distinguishable it is often impossible to say, but in most cases the basal murmur is the louder. When the systolic murmur is loud and rough and best heard at the apex, false diagnoses of "mitral regurgitation" are often made. The murmur is probably due to sclerotic roughening and dilatation of the aortic arch, although it may be loudest at the apex.

When compensation fails, the murmurs often diminish or disappear, and we find one or another type of *gallop rhythm* with short valvular sounds.

Ventricular premature beats ("extrasystoles") and, with increasing decompensation, *absolute arrhythmia* are often present. *Alternation*, permanent, or more often for a few beats following an extra systole, is not at all uncommon. Paroxysmal tachycardia and auricular flutter are occasionally seen.

Delayed A-s–V-s conduction with different degrees of heart block are rather rarely encountered.

For the sure recognition of gross changes in the *myocardium* our present methods of physical examination are always unsatisfactory and often wholly inadequate. Extensive degenerations of the heart wall are not infrequently found at autopsy when there has been no reason to suspect them during life. On the other hand, the autopsy often fails to substantiate a diagnosis of degeneration of the heart muscle, although all the physical signs traditionally associated with this condition were present during life. The following figures from the Massachusetts General Hospital illustrate these difficulties:

Cases of fibrous myocarditis correctly diagnosed....	13 or 22%
Cases of fibrous myocarditis diagnosed in life, but not found post mortem........................	31 or 52%
Cases of fibrous myocarditis found post mortem, but not diagnosed in life..........................	15 or 26%
Total attempts..................................	59

If therefore we are to attempt a diagnosis of myocarditis this must depend upon the history and symptoms of the case; physical examination can sometimes supplement these, sometimes not. Symptoms of *cardiac weakness* developing in a man past middle life, especially in a patient who shows evidences of arterio-sclerosis or high blood pressure, or who has suffered from the effects of alcohol and syphilis, suggest parietal disease of the heart. The possibility is increased if there have been attacks of angina pectoris, Cheyne-Stokes breathing, of heart block or of syncope (Stokes-Adams syndrome).

1. *Summary*

1. The causative factors, the history and symptoms of the case and the condition of other organs are often of more diagnostic value than is the physical examination of the heart itself, which may show little that is abnormal.

2. Among the physical signs, those most reliable are:

(*a*) Increased blood pressure.
(*b*) Sclerosed arteries.
(*c*) Evidences of cardiac hypertrophy and dilatation.
(*d*) Weak, irregular heart sounds.
(*e*) Reduplication of some of the cardiac sounds (gallop rhythm).
(*f*) Murmurs—especially a systolic murmur at the base.
(*g*) Evidences of heart block or of alternation.

2. *Differential Diagnosis*

We have to distinguish the weakened heart of hypertensive cardiovascular disease from—

(*a*) Uncomplicated valvular lesions.
(*b*) Cardiac neuroses.

(*a*) It has been already pointed out that valvular lesions do not necessarily give rise to any murmurs when compensation has failed. Under such circumstances one hears only irregular and weak heart sounds, as in myocardial weakness. The age of the patient, the history of syphilis or "rheumatism," the character of the onset, the presence or absence of arterio-sclerotic manifestations in the brain, kidneys, or extremities, *and above all the blood-pressure measurements*, assist us in diagnosis. High blood-pressure is never found in the "rheumatic" type of heart disease, and in syphilitic aortitis only the systolic not the diastolic pressure is raised. In arterio-sclerotic disease systolic and diastolic pressure are often high. But Allbutt has rightly emphasized the fact that there is a senile or "decrescent" type of arterio-sclerosis without hypertension or cardiac hypertrophy.

(*b*) Weakness and irregularity of the cardiac sounds, when due to "nervous" affection of the heart and unassociated with parietal or valvular changes, is usually less marked after slight exertion. The heart "rises to the occasion" if the weakness is a functional one.

On the other hand, if any serious weakening is present, the signs and symptoms are much aggravated by any exertion.

In "neurotic hearts" the arrhythmia is usually of the juvenile or *sinus* type and the evidence of sexual excess or excessive tobacco can often be found, also the history and signs of a generalized neurosis (see *Cardiac signs of Nervousness*, p. 277).

II. Fatty Degeneration

There are no physical signs by which fatty degeneration of the heart can be distinguished from other pathological changes which result in weakening the heart walls. An extensive degree of fatty degeneration is often seen post mortem in cases of pernicious anæmia, although the heart sounds have been clear, regular, and in all respects normal during life.

III. Senile or "Decrescent" Arterio-sclerosis

When arterio-sclerosis develops in old age, hypertension and cardiac hypertrophy are often absent. This was the case in 58 out of 166 cases of arterio-sclerosis examined by Ophüls[1] (35%); most of these cases were senile. In these cases there may be no complaints at all or only those of angina pectoris. Physical examination shows only—

1. Rough stiff tortuous arteries (especially the brachials).
2. Dilated aortic arch (percussion and *x*-ray).
3. Loud ringing aortic second sound.

The heart and circulation may be otherwise normal.

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[1] Ophüls: Arch. of Int. Med., 1912, Vol. ix, p. 156.

CHAPTER XVI

NEPHROGENOUS HEART DISEASE. GOITRE HEART. CONGENITAL MALFORMATIONS. MISCELLANEOUS CARDIAC LESIONS

I. Nephritic (or Nephrogenous) Heart Disease

From the point of view of physical diagnosis the cardiac enlargement and weakening due to nephritic hypertension presents little to distinguish it from hypertensive cardiovascular disease. Indeed the two are sometimes indistinguishable or simultaneously present. In typical cases, however, we can distinguish nephrogenous heart disease by the following criteria.

1. *The age.*—In 117 cases of weakened heart due to chronic glomerular nephritis (Massachusetts General Hospital) I found the average age 36, while in 93 arterio-sclerotic cardiac cases the age averaged 59 years.

2. *The condition of the urine* (see below, p. 413).

3. *The condition of the arteries.*—Nephritic heart disease often shows thickened arteries, but not often the roughening of calcification.

4. Anæmia, retinitis and "uræmia" are commoner in the nephrogenous than in the arterio-sclerotic type.

The heart itself shows evidences simply of hypertrophy and dilatation as in arterio-sclerosis. There is often gallop rhythm and a basal systolic murmur. Delayed A-s–V-s conduction and heart block are less common than in any other type of cardiac disease.

II. Goitre Heart

Goitres of all sorts, *toxic* and non-toxic, large or small, are apt to be associated sooner or later with high systolic pressure and with some hypertrophy and weakening of the heart. The diastolic pressure is little if at all elevated.

Corresponding presumably with this rise of systolic pressure, it has long been noticed that simple goitres[1] "which produce no other recognizable symptoms, may gradually become associated with marked degenerative changes in the cardiovascular system." The cardiac symptoms come on slowly in the simple non-exophthalmic cases (average time after the appearance of goitre, 14 years in Plummer's series) while in the toxic and exophthalmic cases, cardiac mischief appears within a year.

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[1] Balfour: Mayo Clinic Papers, 1914, p. 371.

The slowly developing cases ("simple goitre") show the worst myocardial weakness, much like that of arterio-sclerotic trouble due to syphilis or other causes, while in the acute toxic cases ("exophthalmic goitre") the cardiac damage is less.

The heart signs, then, may be divided into two groups:

1. In *toxic* (usually exophthalmic) goitre, the heart shows:

(*a*) Enlargement: displacement of the apex to the left and down.

(*b*) Tachycardia.

(*c*) Very loud "angry" sounds.

(*d*) Jumping arteries with bounding then collapsing pulse, often a capillary pulse and a large pulse pressure.

(*e*) In later stages absolute arrhythmia (auricular fibrillation).

2. In the "*simple*" and usually *non-toxic* group there gradually develop signs of myocardial weakness not distinguishable from those associated with arterio-sclerosis. We find a large, irregular heart with systolic murmurs at all the orifices and usually a high systolic pressure. Diastolic pressure may or may not be elevated. The peripheral arteries often show sclerosis.

III. Congenital Heart Disease

Dr. Maude E. Abbott[1] considers *pulmonary stenosis* as "probably the commonest of all (congenital) cardiac anomalies," but the cases are reported in so scattered a way that exact summation is difficult. Her tables show the following main types among 631 cases analyzed:

1. Stenosis of the descending aortic arch (coarctation of the aorta)	212;	with other defects in	119.
2. Defective interventricular septum	189;	pulmonary stenosis in	75.
		other defects in	68.
3. Patent ductus arteriosus	166;	with other defects in	102.
4. Pulmonaty stenosis	116;	with other defects in	107.
5. Widely patent foramen ovale	89;	with other defects in	71.
6. Transposition of the arterial trunks	70;	with other defects in	13.
7. Hypoplasia of the aorta	46;	with other defects in	44.

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[1] Osler's Modern Medicine, Vol. IV, p. 323.

These anomalies may be divided into two groups:

(*a*) *Those with marked cyanosis*—pulmonary stenosis, transposition of the arterial trunks and in a few cases of septum defect.

(*b*) *Those with little or no cyanosis*—aortic coarctation, most septum defects, patent ductus arteriosus, and hypoplasia of the aorta.

1. *Pulmonary Stenosis*

A harsh systolic murmur (and in 15 per cent. a systolic thrill) is recognized at the base of the heart and usually faint or absent at the apex. Cyanosis is usually marked. In most cases these signs are best marked in the pulmonary area, in or above the second left interspace, but the murmur is often audible to some extent all over the chest except in the back.

The heart is enlarged to the right and upwards, and its impulse may bulge the precordia and sometimes shakes the head and chest. The pulmonic second is usually weak or absent but may be actually increased.

If a thrill is absent there is probably an associated defect in the interventricular septum.

2. *Stenosis of the Descending Aortic Arch* (*Aortic Coarctation*)

There may be no signs or symptoms. In a few cases the blood supply to the head and arms is too great; that to the rest of the body, too small. Hence there is headache, tinnitus, insomnia, suffusion of the head and neck. As a rule, symptoms appear late in life and under special strains (*e.g.*, endocarditis).

Physical Signs.—There may be violent pulsation of the subclavians and carotids with dilated tortuous vessels over the chest. Thus a collateral circulation between the arteries above the stenosis and those below it was established in at least 66 of 142 adult cases in Dr. Abbott's collection. The pulse in the abdominal aorta and femorals is weak. Hypertrophy and dilatation of the whole heart occurred in 87 of 142 cases.

In some cases no murmur is present. In others a loud systolic is heard at the base or apex.

3. *Patent Ductus Arteriosus*

In infants the signs are not distinguishable from those of septal defects. Thrill was present in 17 of 64 cases (Abbott's series), usually in the second left space. It is sometimes systolic, sometimes continuous through the whole cardiac cycle. The murmur may likewise be systolic or continuous—rarely divided into systolic and diastolic. Among 46 adult cases it was systolic in 19, continuous in 14, "double" in 9.

The murmur is often extraordinarily loud and harsh.

The second sound may be increased, diminished, or absent.

4. *Defective Interventricular Septum*

Physical signs may be absent and cyanosis is often absent. A systolic thrill is present in about one-third of the cases, diffused over the precordia or with maximum intensity at the base. There may be precordial pulsation and bulging.

A loud systolic murmur is the commonest sign and is present in about two-thirds of the cases. Rarely a diastolic murmur is also heard. It is widely diffused but loudest in the pulmonary area or just below it. It may last through the entire cardiac cycle with a systolic accent as in patent ductus arteriosus.

5. *Defective Interauricular Septum (Foramen Ovale)*

Signs are often absent and even cyanosis is rare. When signs are present they are like those of ventricular defects, except that the murmur is often heard in the left back and is sometimes presystolic in time. Both murmur and thrill vary in intensity and in time with the position of the patient (Abbott).

6. *Transposition of the Arterial Trunks*

Marked cyanosis with clubbed fingers may be the only sign in uncomplicated cases. Even this may be absent and the only suggestion of the lesion may be an accented pulmonic second sound with evidence of hypertrophied right ventricle. These features *plus* cyanosis and *no murmurs* may suggest the diagnosis.

7. *Hypoplasia of the Aorta*

Obstinate anemia in an adolescent with a cardiac hypertrophy and dilatation for which no cause is found, are the only known signs of the disease. Diagnosis is usually impossible.

IV. OTHER DISEASES WHICH WEAKEN THE HEART

1. *Acute Myocarditis*

The myocardium is seriously, though not incurably, affected in all continued fevers, owing less to the fever itself than to the toxæmia associated with it. "Cloudy swelling," or acute degeneration of the muscle fibres, is produced by "common colds," tonsilitis and other relatively mild infections, while a general septicæmia due to pyogenic organisms may produce extensive degeneration of the heart within a few days.

The *physical signs* are those of *cardiac weakness*. The most significant change is in the quality of the first sound at the apex of the heart, which becomes gradually shorter until its quality is like that of the second sounds, while in some cases its feebleness makes the second sounds seem accented by comparison. Soft blowing systolic murmurs may develop at the pulmonary orifice, less often at the apex or over the aortic valve.

The apex impulse becomes progressively feebler and more like a tap than a push. *Irregularity* and *increasing rapidity* are ominous signs which may be appreciated in the radial pulse, but still better by auscultation of the heart itself. In most of the acute infections, evidence of dilatation of the weakened cardiac chambers is rarely to be obtained during life (although at autopsy it is not infrequently found), but in *acute articular rheumatism* an acute dilatation of the heart appears to be a frequent complication, independent of the existence of any valvular disease. Attention has been especially called to this point by Lees and Boynton (*British Med. Jour.*, July 2, 1898) and by S. West.

Influenza is also complicated not infrequently by acute cardiac dilatation and followed by symptoms of cardiac weakness or by angina pectoris.

V. Miscellaneous Affections of the Heart

1. *Tachycardia (Rapid Heart)*

Simple quickening of the pulse rate, or tachycardia, which may pass altogether unnoticed by the patient himself, is to be distinguished from palpitation, in which the heart beats, whether rapid or not, force themselves upon the patient's attention.

The pulse rate may vary a great deal in health. A classmate of mine at the Harvard Medical School had a pulse rarely slower than 100, yet his heart and other organs were entirely sound. Such cases are not very uncommon, especially in women. Temporarily the pulse rate may be greatly increased, not only by exercise and emotion, but by the influence of fever, of gastric disturbances, or of the menopause. Such a tachycardia is not always of brief duration.

The effects of a great mental shock may produce an acceleration of the pulse which persists for days or even weeks after the shock.

Among organic diseases associated with tachycardia and weakening of the pulse, the commonest are those of the heart itself. Next to them, Graves' disease (thyrotoxicosis) and functional neuroses (some of them sexual) are the most frequent causes of tachycardia.

2. *Cardiac Symptoms of Nervousness*

(Effort syndrome, neuro-circulatory asthenia, irritable heart of soldiers, D. A. H.)

People of nervous temperament (congenital) go to pieces under strains which the average person bears without disturbance. War brings this defect into prominence—often for the first time. But careful questioning usually proves that college examinations, athletic stunts and other exciting experiences, have produced symptoms similar to those which in the soldier under battle strain prove disabling. (So it was in 47 out of 50 cases studied by Friedlander and Freyhof, Arch. of Int. Med., Dec., 1918.)

Such people may give out with digestive, respiratory or urinary symptoms (vomiting, dyspnœa or polyuria), but most often the patient's attention (and that of his physician) is directed to his heart by the following symptoms:

1. *Tachycardia* and palpitation.
2. Cardiac *pain* or distress.
3. *Dyspnœa* on emotional or physical strain.
4. *Weakness*, vertigo and fainting.
5. *Tremor*, flushing and sweating.

Tobacco often intensifies these symptoms.

Physical examination shows:

(*a*) Very loud, abrupt and rapid heart action without any other significant alterations.

(*b*) Moderate elevation of systolic blood pressure, while diastolic pressure remains normal or nearly so.

(*c*) Deficient power of muscular work without exhaustion.

(*d*) Trembling, clammy, cyanotic hands.

(*e*) Precordial tenderness in 48% (Lewis).

Systolic murmurs are often heard at the valve-areas. Something often taken for a systolic thrill is occasionally felt at the apex. There is no enlargement and usually no irregularity of the heart. *X*-ray and electrocardiography show nothing of significance.

The most important duty of the physician is to convince first himself and then his patient that the heart is perfectly sound and that the only trouble with him is his nervousness. Great harm may be done by a doubtful or mistaken diagnosis. Emphatic reassurance based on careful examination is the essential.

Hyperthyroidism is suggested in many of these cases by the tachycardia, tremor, nervousness and sweating. But the lack of any change in the basal metabolism, as shown by the studies of F. W. Peabody, makes thyroid involvement very improbable.

Psychic and physical training improves many cases. Digitalis and rest are useless or harmful.

3. *Post Infectious Tachycardia*

A condition almost precisely like that just described sometimes occurs during convalescence from pneumonia, typhoid and other infections. Usually this occurs in those of congenital neurotic type. Recovery is more prompt and lasting than in the non-infectious cases.

4. *The Supposed Effect of Tobacco*

The disturbance produced on the heart by tobacco has been greatly exaggerated. There is no cardiac abnormality or group of abnormalities that can be referred to the effects of tobacco alone. Persons constitutionally subject to tachycardia, palpitation or to occasional premature contractions may find these symptoms exaggerated or brought into the foreground by the abuse of tobacco. But almost invariably one finds other factors in the background—neurotic temperament, bad hygiene, sexual excess, convalescence from infectious disease.

Physical examination shows either (*a*) the signs described under the "Cardiac Symptoms of Nervousness" (p. 277) or (*b*) more or less frequent premature beats.

5. *"Acute Dilatation of the Heart," "Athlete's Heart"*

If recent acute infections and arterio-sclerosis are excluded, we can say that *there is no reason to believe that acute dilatation or athlete's heart exists.* After athletic contests and Marathon races, the heart does not dilate. Indeed, *x*-ray shows that it is generally slightly smaller than usual.

Hypertrophy does not, as far as I can ascertain, result from muscular exertion, no matter how great and prolonged—when arteriosclerosis and syphilis can be excluded.

The cases of weak, rapid or irregular heart action, occurring in adolescence or later and wrongly supposed to be due to athletics, are in fact cases of nervous heart or post infectious weakness, accentuated often by the alarm produced by a mistaken diagnosis of heart strain, dilatation or athlete's heart. Of course in cases of organic heart disease, valvular or myocardial, compensation may be broken by excessive exercise.

6. *Bradycardia* (*Slow Heart*)

In many healthy adults the heart seldom beats over 50 times a minute.

1. Among the causes which may produce for a short time an abnormally slow heart-beat are:

(*a*) *Exhaustion;* for example, after fevers, after parturition, or severe muscular exertion.

(*b*) *Toxæmia;* for example, jaundice, uræmia, auto-intoxications in dyspepsia.

(*c*) In certain *hysterical and melancholic* states and in *neurotic children*, the pulse may be exceedingly slow.

(*d*) An *increase of intracranial pressure*, as in meningitis, cerebral hemorrhage, depressed fracture of the skull. Possibly in this category belong the cases of bradycardia sometimes seen in epileptiform or during syncopal attacks. Bradycardia from any one of these causes is apt to be of comparatively short duration.

7. *Heart Block*

(See above, page 116.)

8. *Palpitation*

Best defined as an "irregular or forcible heart action *perceptible to the individual.*" The essential point is that the individual becomes conscious of each beat of his heart, whether or no the heart action is in any way abnormal.

(*a*) In irritable conditions of the nervous system, such as occur at puberty, at climacteric, or in neurasthenic persons, palpitation may be very distressing. Temporary disturbances, such as fright, may produce a similar and more or less lasting effect.

(*b*) The effect of high altitudes, or of even a moderate elevation (1,500 feet) is sufficient to produce in many healthy persons a quickening and strengthening of the heart's action, so that sleep may be prevented. After a few nights this condition usually passes off, provided the heart is sound.

(*c*) Abuse of tobacco and tea have a similar effect.

(*d*) *Any marked arrhythmia*, with or without organic lesions, may be noticed as "palpitation" by the patient.

(*e*) *Heart block* (see p. 116).

Auscultation of a palpitating heart shows nothing more than unusually loud and ringing heart sounds, but since palpitation is often associated with arrhythmia of one or another type we must be careful to exclude the palpitation symptomatic of acute exhaustion of the heart, such as may occur in debilitated persons after violent or unusual exertion. In this condition dyspnœa upon slight exertion becomes marked. (See above—Cardiac Symptoms of Nervousness.) It goes without saying that in almost any case of organic disease of the heart, palpitation may be a very marked and distressing symptom.

CHAPTER XVII

DISEASES OF THE LUNGS

BRONCHITIS, PNEUMONIA, TUBERCULOSIS

I. Tracheitis

In connection with bronchitis or as a forerunner thereof, inflammation of the trachea is not uncommon. It gives rise to no characteristic physical signs, but is to be suspected when the patient complains of *cough with pain over the upper portion of the sternum.*

II. Bronchitis

1. *Acute Bronchitis*

Inflammation of the larger bronchial tubes is not often the cause of any definite physical signs, but with every paroxysm of coughing the patient may feel pain in an area corresponding exactly to the anatomical position of the primary bronchi. I have seen patients indicate most accurately the situation of the large tubes when pointing out the position of pain produced by coughing.

In the vast majority of cases of *acute bronchitis, foci of bronchopneumonia are also present*, but the physical signs are usually those of an inflammation of the smaller bronchi, and the swelling of their walls, with or without exudation, which is manifested as follows:[1]

(1) Diminution in the intensity of vesicular breathing over the area affected (rarely in the earliest stages the breath sounds are exaggerated and harsh, especially in the upper portions of the chest).

(2) *Râles*, fine crackles or squeaks especially in the lower backs or axillæ in adults. In children the râles are more widely distributed and are often musical or squeaking over bronchi which are narrowed without any considerable amount of exudation, as is the case in the earliest stages of many cases, and bubbling, crackling, or clicking in later stages, when watery or viscid exudation is present in the tubes. The calibre of the bronchi affected can be estimated from the coarseness or fineness of the râles. Low-pitched groaning sounds point to a

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[1] Bronchitis may exist without râles, but cannot be diagnosed without them. Occasionally they are present only in the early morning.

stenosis of a relatively large bronchus, while squeaking and whistling sounds are usually produced in the smaller tubes. Large, bubbling râles are much less often heard than the finer, crackling variety. The latter are produced in the smallest tubes, the former in the larger variety.

Simple non-tuberculous bronchitis is almost invariably *bilateral* or symmetrical, and affects most often the lower two-thirds of the lungs, leaving the apices relatively free. It is almost never confined to an apex. When râles are to be heard on one side of the chest only, and when they persist in the same spot for days and weeks, tuberculosis is always to be suspected, especially if the râles are localized at the summit of one or both lungs. At the base, such a patch is usually non-tuberculous. It should never be forgotten that the tubercle bacillus is capable of exciting a bronchitis indistinguishable from other varieties of bronchitis, except by its tendency to show itself at the apex of the lung and on one side only; most cases of pulmonary tuberculosis begin in this way.

The only other variety of bronchitis which is often unilateral is that due to the influenza bacillus. In the course of a case of influenza, a unilateral localized bronchitis not infrequently occurs. Over a patch of lung, perhaps the size of the palm of the hand, fine, moist râles may persist for weeks, finally clearing up only after the patient has resumed his ordinary occupation. Doubtless such localized patches of bronchitis are accompanied by foci of lobular pneumonia too small to be detected by our present methods of physical examination. Sometimes they can be demonstrated by the *x*-ray.

Percussion dulness is absent in bronchitis except near the end of fatal cases, when the lung is stuffed with mucus and pus, or when atelectasis has occurred owing to extensive plugging of the larger bronchi. These events are rarely seen, and in general, the negative results of percussion are of great value in excluding solidification or fluid exudation.

Occasionally percussion resonance may be increased owing to a slight temporary overdistention of the air vesicles from coughing.[1]

Inspection usually shows little or nothing of diagnostic importance in acute bronchitis. Long-standing cases, complicated as they almost invariably are by emphysema, present changes in the shape of the thorax; but these are due to the emphysema rather than to the bronchitis. In children, acute bronchitis sometimes involves so many of the smaller bronchi that dyspnœa and use of accessory muscles of respiration are notable. But this usually means atelectasis, broncho-pneumonia, or laryngeal spasm, in addition to the bronchitis.

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[1] In children examined during a crying-spell, a cracked-pot sound can usually be elicited by percussion. This is in no way characteristic of bronchitis and can often be obtained in healthy infants.

From violent coughing the jugulars may be distended, but no systolic pulsation occurs in them.

Voice sounds and tactile fremitus are sometimes increased owing to the coincident broncho-pneumonia. In other cases they are normal or decreased.

(a) *Differential Diagnosis*

Œdema of the lung and bronchial asthma are the only pathological processes (except hemorrhage into the lung substance) which give rise to signs like those of bronchitis.

(1) In *œdema of the lung*, or in pulmonary apoplexy, one may find, as in simple bronchitis, a diminished vesicular breathing with crackling râles, but œdema of the lung is almost always best marked in the dependent portions; that is, in the posterior parts of the lung if the patient has been lying upon the back, or in the lower lobes if he has been sitting up. Dulness and diminished tactile fremitus are usually present, though slight. The recognition of a cause for the œdema, for example a non-compensated heart lesion, and the absence of fever or leucocytosis materially aid the diagnosis.

(2) *Bronchial asthma* or spasm of the finer bronchi gives rise to squeaking and groaning sounds similar to those heard in the earlier stages of many cases of bronchitis. But in bronchial asthma, fever is usually absent, the râles are chiefly *expiratory*, and expiration is prolonged and intensified. Moreover, the inhalation of a few drops of amyl nitrite will temporarily dispel râles due to bronchial spasm, while on the râles of dry bronchitis it has no effect (Abrams).

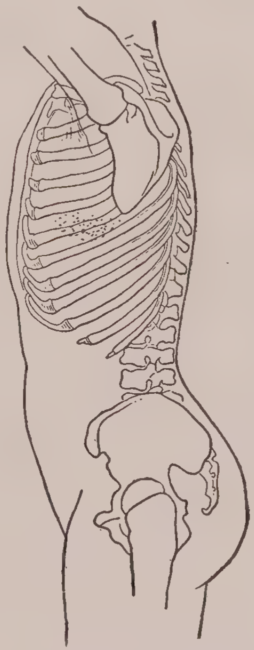

FIG. 173.—The Dots are Placed over the Area where Atelectatic Crepitation is Oftenest Heard.

(3) *Broncho-pneumonia.* In many cases of lobular or broncho-pneumonia the physical signs are exclusively those of the coexisting

bronchitis. In such cases the diagnosis of bronchitis is not wrong, but does not cover the whole ground. Indeed I am doubtful whether it ever does. There is, to my mind, no proof that acute bronchitis without broncho-pneumonia ever exists.

(4) *Muscle sounds.* Under certain circumstances (cold, nervousness), the rumbling noises produced by muscular contractions in the chest wall may simulate râles so closely that the diagnosis of bronchitis may be strongly suggested. The differentiation between râles and muscle sounds has already been discussed (see above, p. 150).

(5) *Atelectatic crepitation.* Crackling râles heard over the thin margins of the lungs at the base of the axilla or along the edges of the manubrium are often due to atelectasis (see above). From bronchitis they are distinguished by their situation and by the lack of symptoms. They are best heard at the point shown in Fig. 173 on page 283.

2. *Chronic Bronchitis*

So far as the bronchitis itself is concerned, there may be no difference in the physical signs between the acute and chronic forms of the disease; but in the latter, one almost invariably finds associated with the bronchitis itself a considerable degree of cardiac weakness with emphysema and asthma. Indeed, the foreground of the clinical picture and the bulk of the physical signs are made up by these three diseases, rather than by the bronchitis itself. The more we study bronchitis the more it vanishes from sight as a clinical entity. "*Acute bronchitis" is fast turning into acute broncho-pneumonia; "chronic bronchitis" is being recognized as chronic pulmonary œdema from weak heart action.* Lord has analyzed 161 autopsied cases in which the history and physical signs were such as are usually attributed to "chronic bronchitis" and concluded that the correct diagnoses were as follows:

Heart weakness,	103	Chronic pneumonitis or abscess of the lung,	15
Neoplasm of lung,	5	Other conditions,	7
Tuberculosis,	31		
			Total = 161

It seems very doubtful whether "chronic bronchitis" exists at all except as a minor element in other diseases.

III. Croupous Pneumonia

In its typical form croupous or fibrinous pneumonia produces solidification of one or more lobes, usually the lower, the process being accurately bounded by the interlobular fissures. Although the physical signs of the earlier stages differ considerably from those of the later ones, there seems to be no sufficient ground for marking off stages of engorgement and of red and gray hepatization, for *clinically* these stages cannot be distinguished.

The solidification may begin in the deeper parts of the lung ("*central pneumonia*"), so that no physical signs are obtainable unless those shown by *x*-ray, or unless the process extends to the surface of the lung. But in many so-called "central pneumonias" there is really no localization or solidification at all. The process is an acute general pneumococcus infection which may or may not settle in the lung.

Massive pneumonia, in which the bronchi as well as the air cells are plugged with fibrin and leucocytes, is a relatively rare form of the disease, but possesses great clinical importance on account of the marked resemblance between its physical signs and those of pleural effusion.

The right lung alone is involved in 54 per cent of cases, the left alone in 38 per cent., both lungs in 8 per cent. (Jürgensen's analysis of 6666 cases quoted by Lord). In 70 per cent. of cases the lower lobe is affected.

1. *Physical Signs*

(*a*) *Inspection.*—The aspect of the patient frequently suggests the diagnosis; the face is anxious, often flushed or slightly cyanosed, the flush sometimes affecting most strikingly the side of the face corresponding to the lung affected.[1] Herpetic vesicles ("cold sores") are often to be seen around the mouth or nose. The rapid, difficult breathing is at once noticeable, and expiration is often accompanied by a grunt. The use of the accessory muscles of respiration and the dilatation of the nostrils attract attention.

The combination of marked dyspnœa with absence of dropsy is met with more frequently in pneumonia than in any other disease. Both sides of the chest usually move alike, but occasionally the affected side shows deficient expansion, especially in the later stages of the disease, and the other side of the chest shows increased respiratory movements (compensatory). Rarely the pulsations of the heart may be transmitted to the chest wall through the affected lung.

When pneumonia attacks a feeble old man, or follows injuries (surgical pneumonia), its onset may be insidious, and none of the phenomena just described may be seen.

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[1] Probably because the patient is apt to lie upon the affected side.

(*b*) *Palpation.—In the great majority of cases tactile fremitus is markedly increased over the affected area,*[1] but in case the bronchi are occluded by secretions or fibrinous exudate, fremitus may be diminished or altogether absent. A few hard coughs will sometimes clear out the tubes and thus materially assist the diagnosis. Occasionally some muscular spasm or an increase in superficial temperature of the affected side may be noticed by palpation, and rarely one feels a friction rub due to the pleurisy which accompanies the disease.

(*c*) *Percussion.—Over the area affected, the percussion note is generally dull and may be almost flat,* except in the earliest and latest stages of the disease, in which it may have a tympanitic quality with or without slight dulness. More marked tympany is usually present over the unaffected lobes of the diseased lung (that is, over the upper lobes in most cases) and at the base of the sound lung (Lord).

The conditions just described represent the great majority of cases, but the following exceptions occur:

(1) In the pneumonias of children, and occasionally in adults, dulness may be absent.

(2) When the lower lobe of the left lung is affected, a distinctly tympanitic quality may be transmitted to the consolidated area from a distended stomach or colon.

(3) In central pneumonia there may be no change in the percussion note, or it may be unusually full and deep so that the sound side seems dull by comparison.

A solidified lobe increases so much in size that the area of dulness corresponding to it often seems incredibly large. Thus, although the lower lobe reaches in health not more than half-way up the scapula, when solidified it produces dulness throughout nearly the whole back.

The right base is the most frequent seat of pneumonic solidifications, but the dulness corresponding to it is often first noticeable in the posterior axillary line. A dulness appreciable only in the front of the chest is almost sure to correspond to the upper lobe, while signs in the lower part of the right axilla correspond to the middle lobe. Many cases of central pneumonia first appear at the surface in one or the other axilla.

As regards the amount of solidification needed to produce percussion dulness, Wintrich says that the minimum is a patch 5 cm. in diameter, 2 cm. deep, and superficially situated.

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[1] By using the edge instead of the flat of the hand the boundaries of solidified lobes may often be very accurately marked out by means of the tactile fremitus.

Percussion often makes us aware of an increased (perhaps muscular) resistance or diminished elasticity of the affected side, although the resistance is seldom as marked as in large pleural effusions.

(*d*) *Auscultation.*—In the great majority of cases typical tubular breathing is to be heard over the affected area. Since a whisper is practically a forced expiration, this *tubular quality* is very well brought out if the patient is made to whisper "one, two, three," or any other succession of syllables, and by this method the fatigue and pain of deep breathing may be saved. By this use of the whispered voice, one may accurately mark out the boundaries of the consolidated area without tiring the patient, and may demonstrate in many cases that it coincides with the boundaries of one lobe of the lung.

In the earliest stages of the disease the breathing may be broncho-vesicular; more often it is *feeble* or suppressed over the consolidated area, and fine crackling râles may be heard at the end of inspiration. The crepitant or fine crackling râle is more often heard at the onset of broncho-pneumonia than of lobar types.[1]

If the smaller bronchi are blocked or if the pleura is much thickened, as is not infrequently the case, respiration is absent or very feeble; such cases may be mistaken for pleuritic effusion.

In cases of "central pneumonia," that is, when the area of solidification is in the interior of the organ, there may be no change in the breath sounds, or *a bronchial element may be faintly audible on auscultation with the unaided ear, and only by this method.*

The intensity of the spoken or whispered voice is greatly increased over the area of consolidation, and sometimes the words can be distinguished. The nasal twang known as "*egophony*" is occasionally to be heard. In the majority of cases, as has been already stated, the right lower lobe posteriorly is affected, so that the consolidated area is immediately in apposition with the spinal column. Under these circumstances, it is not at all uncommon to hear bronchial breathing transmitted from the consolidated lobe to a narrow zone close along the spinal column on the *sound* side. Such a zone is often mistaken for consolidation.

The signs are usually less marked in the axilla and in the front of the lung, but in a minority of cases, and especially when the upper lobes are affected, the signs are wholly in the front. When searching for evidences of consolidation in persons suspected to have pneumonia, one should never omit to examine the apices and *very summit* of the armpit, pressing the stethoscope up behind the anterior fold of the axilla.

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[1] Crepitant râles are rarely heard in the pneumonias of infancy and old age. They are not peculiar to pneumonia, but occur in pulmonary œdema or hemorrhagic infarction—conditions easily distinguished from pneumonia.

In examining the posterior lobes, when the patient is too weak to sit up and is loath even to turn upon the side, the Bowles stethoscope is a great convenience, owing to the ease with which its flattened extremity may be worked in between the patients and the bed-clothes without causing any discomfort. *X*-ray may be of important service in the detection of *central* pneumonia. The shadow of a solidified patch is mottled and ill-defined in comparison with pleural effusion or tumor.

When *resolution* begins, the signs may almost completely disappear within a few hours. More frequently the bronchial breathing is modified to broncho-vesicular, dulness and bronchophony become less marked, fine crackling râles (crepitans redux) or coarser moist bubbles appear, and the lung gradually returns to its normal condition within a period of three to eight days. In the most active stages of the disease the entire absence of râles is the rule. In most cases the solidification of the lung persists after the fall of the temperature; indeed, it may be weeks or even months before it clears up, and yet the lung may be perfectly sound in the end. On the other hand, abscess or gangrene may develop in the solidified lobe. This was the case in 2 per cent. of 500 cases analyzed by Lord. In the 153 autopsies of Stone, Bliss & Phillips (see p. 289) pulmonary abscess occurred in 26 or 16 per cent. Organization or fibrosis of the affected lobe was

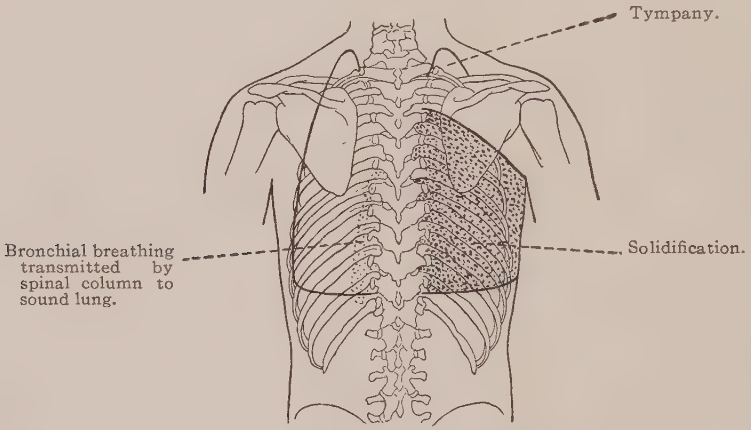
Tympany.
Bronchial breathing transmitted by spinal column to sound lung.
Solidification.

FIG. 174.—Diagram of Signs in Pneumonia.

found in 7.6 per cent. of 210 autopsied cases of pneumonia of the Massachusetts General Hospital. Small serous effusions were found in 26 per cent. of the same series. Commonest and most important, however, is the *post-pneumonic empyema* (basal or interlobar) which is *often mistaken for delayed resolution.* The latter is rare; empyema is discovered in about 4 per cent. (see below, p. 330).

"*Wandering pneumonia*" is a term applied to cases in which the consolidation disappears in one lobe only to reappear in another, or spreads gradually from lobe to lobe. The physical signs in such cases do not differ essentially from those already described.

Complications.—Endocarditis is found in about 1 per cent. of all cases and 4 to 7 per cent. of fatal cases. Pericarditis is found in from 13 per cent. (Musser and Norris) to 33 per cent. (Stone, Bliss & Phillips, Arch. of Int. Med., Oct., 1918) when autopsied cases only are studied. Forty per cent. or more of these cases of pericarditis are not recognized in life; acute purulent peritonitis was demonstrated in 5 or 3.2 per cent. of 154 cases autopsied at the Massachusetts General Hospital. Stone, Bliss & Phillips found it in 24 out of 153 autopsied cases (15 per cent.). Meningitis complicated 2.5 per cent. of the same series. Otitis media was found in 3.4 per cent., acute arthritis in .4 per cent. Peripheral venous thrombosis was seen in 10 of 949 cases (1 per cent.) studied by Sears and Larrabee.

2. *Summary*

In a typical case one finds (oftenest at the right base behind)

1. Dulness on percussion.
2. Increased tactile fremitus and voice sounds.
3. Tubular breathing and occasionally crepitant râles.

These signs occurring in connection with fever, cough, rusty sputa, pain in the side, dyspnœa, herpes and leucocytosis are sufficient for the diagnosis.

But many cases are not typical when first seen. The following are the commonest anomalies:

(*a*) There may be tympany instead of dulness, especially in children or when the solidification is at the left base.

(*b*) The breathing may be *feeble* but vesicular in character, or it may be absent, in case bronchi are plugged; from the same cause. In early stages fine crackles with feeble vesicular breathing and slightly increased voice sounds is the commonest combination.

(*c*) Tactile fremitus may be diminished.

A hard cough may clear out the bronchi and produce a sudden metamorphosis of the physical signs, with a return to the normal type.

In these atypical cases, we have to fall back upon the symptoms, the history, the blood, and sputa for help in the diagnosis.

Deep-seated pneumonic processes may appear at the surface in out-of-the-way places, *e.g.*, at the summit of the axilla, and the area of demonstrable physical signs may be no larger than a silver dollar. A thorough examination of every inch of the chest and an *x*-ray study are therefore essential in doubtful cases.

In the later stages of the disease, crepitant or other fine râles often appear, and the signs of solidification suddenly or gradually disappear.

3. *Differential Diagnosis*

Pneumonic solidification is to be distinguished from

(1) Pleuritic effusion, serous or purulent.
(2) Tuberculosis of the lung.
(3) Compression of the lung.

(1) From serous effusion, pneumonia is to be distinguished in the great majority of cases by differences in the onset, course, and general symptoms of the disease. In pneumonia the patient is far more suddenly and violently attacked, the dyspnœa is much greater, cough and pain are more distressing and more frequent, the temperature is higher, and the sputum often characteristic. In pleuritic effusion the dulness is usually more intense than in pneumonia. Tactile fremitus and voice sounds are increased in pneumonia (except when the bronchi are plugged); decreased or absent in pleuritic effusion. Bronchial breathing may be heard in both diseases, but is usually feeble and distant when occurring in pleurisy, and loud in pneumonia. *If the affection be on the left side, the diagnosis is much aided by the presence of dislocation of the heart*, which is produced by pleuritic effusion and never by pneumonia. In cases of pneumonia with occluded bronchi one may have every sign of pleuritic effusion—flatness, absent breathing, voice and fremitus—and in such cases the absence of any dislocation of the heart, provided the disease is upon the left side, is very important. If a similar condition of things occurs upon the right side, one may have to fall back upon the symptoms and upon such evidence as the blood count, herpes, sputum, etc. The above applies to serous pleurisy—tuberculous or streptococcic ("rheumatic"). Pneumococcus empyema on the other hand, especially when it accompanies or follows pneumonia (as it almost always does), is in its early stages almost or quite impossible to distinguish from pneumonia.

Exploratory puncture is often the only method of diagnosis and should be *practiced in every doubtful case.* Aside from puncture the most reliable signs of fluid are:

1. Flatness (not dulness) on percussion.
2. Absent tactile fremitus.
3. Displaced heart (especially in left sided empyema).
4. *X*-ray examination.

(2) Tuberculosis of the lung causing, as it may, a diffuse solidification of the organ, may be indistinguishable from pneumonia if we take account only of the physical signs, but the two diseases can usually be distinguished without difficulty by the difference in their symptoms and course, and by the presence or absence of tubercle bacilli in the sputum.

(3) *Compression* of the left lung by a pericardial effusion and of either lung by some subdiaphragmatic lesion (perinephric abscess, hepatic abscess) often simulates pneumonia; indeed the physical signs may be indistinguishable. Diagnosis depends mostly on establishing the presence or absence of a *cause* for such compression. The *x*-ray demonstration of an abnormally elevated diaphragm may be of great value.

(4) *Pulmonary infarction or thrombosis* is often mistaken for pneumonia. Correct diagnosis depends not on physical signs but on recognizing a possible *cause* for such infarction, *i.e.*, typhoid fever, failing cardiac compensation, peripheral thrombosis, childbirth with or without sepsis, surgical operations. The fever usually appears later than in pneumonia. Bloody sputa is commoner in infarction than in pneumonia. The signs are

1. A patch of pleural friction or of crackling râles, often disappearing without any other signs in 2–3 days.
2. A small patch of consolidation exactly like pneumonia lasting 3–4 days.
3. Signs like those of pleural effusion but with no fluid obtainable on tapping.

In all "queer pneumonias" infarct should be suspected.

Often the causative peripheral venous thrombosis is unsuspected until bloody sputa (pulmonary infarct) reveal its results.

IV. INHALATION PNEUMONIA. ASPIRATION PNEUMONIA

When food or other foreign substances are drawn into the air passages, as may occur, for example, during recovery from ether narcosis, a form of broncho-pneumonia may be set up, in which the solidified patches are not infrequently large enough to be recognized by the ordinary methods of physical examination.

The lesions are usually *bilateral* and accompanied by a general bronchitis. Slight dulness and indistinct bronchial breathing can usually be made out over an irregular area in the backs of both lungs.

The signs are considerably less marked than in croupous pneumonia, and the boundaries of the irregular patches of disease do not correspond to those of a lobe of the lung.

If not rapidly fatal, the disease may be complicated by pulmonary gangrene or abscess and large quantities of fetid pus may be spit up.

V. Broncho-Pneumonia (Catarrhal or Lobular Pneumonia)

1. *Acute Broncho-Pneumonia*

Multiple small areas of solidification scattered through both lungs, interspersed with areas of collapse, and usually associated with diffuse bronchitis, occur very frequently in children, producing severe dyspnœa, cyanosis, cough, and somnolence, and running a very fatal course.

The solidified lobules may fuse so as to form considerable areas of hepatized lung, or there may be no lesion larger than a pea.

This is the usual type of "lung fever" in infants, although ordinary lobar pneumonia is fully as common in older children. Of 195 cases found at autopsy in Lord's series only 14 were in children (since the hospital receives very few children). Suppurative peritonitis was the commonest cause (35 cases). Other infectious diseases and a variety of debilitating conditions (cancer, nephritis, brain tumor, etc.) make up the other causes.

The widespread atelectasis of the lower lobes which is associated with the disease in most cases owing to the plugging of the bronchi with tenacious secretions, is probably as serious in its effects as the pneumonic foci themselves.

The anterior and upper parts of the lungs often become distended with air (vicarious emphysema) and render the physical signs very confusing and deceptive. Much milder and less widespread types of broncho-pneumonia often run their course under the name of "acute bronchitis" and are not discovered unless the patient happens to die of some other cause. The physical signs are merely those of bronchitis and often the patients do not even take to bed.

(*a*) *Physical Signs*

In the majority of cases there are no physical signs except by *x*-ray, and the diagnosis has to be made largely from the symptoms and course of the disease. The consolidated areas are usually too small to give rise to any dulness on percussion, or to any change in the voice sounds, or fremitus, so that auscultation shows, as a rule, nothing more than *patches of fine râles occurring at the end of expiration*. Careful listening usually reveals a diminished respiration as well, but this is not a "showy" sign and most physicians notice only the râles and call the case "bronchitis." Localized tympanitic resonance is sometimes present over the diseased area, making the sounder portions of the lungs seem dull by comparison. Occasionally, when many lobules have fused into a single mass of larger area, the ordinary signs of consolidation may be obtained, although they may disappear within twenty-four or forty-eight hours and appear in another situation. As above said, the diagnosis is usually to be made, if at all, from the combination of the *physical signs of a localized bronchitis with the symptoms*

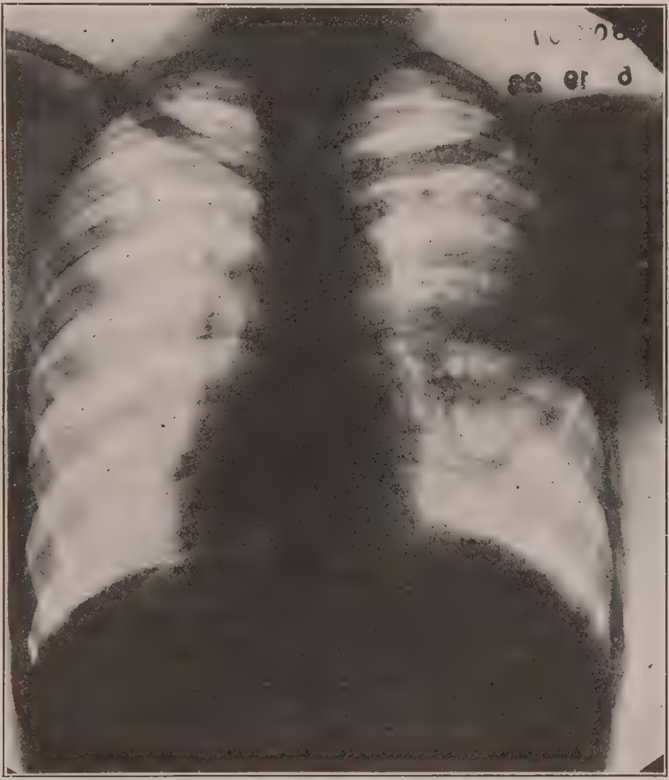

FIG. 175.—Pneumonia—right lung. Note wedge shaped area with its base in the right axilla.

of pneumonia. "This patient," we say, "has only the signs of bronchitis, but he is too sick. The cyanosis, dyspnœa, and fever are too marked. He is sicker than simple bronchitis will account for, but I am in doubt whether there is any such thing as 'simple bronchitis.' It seems to me probable that what has usually been called 'simple bronchitis' with patches of râles here and there is, in fact, a relatively mild type of broncho-pneumonia with some associated bronchitis."

(*b*) *Differential Diagnosis*

(*a*) *Acute pulmonary tuberculosis may* be indistinguishable from broncho-pneumonia by the physical signs alone. But in the vast majority of cases tubercle appear at the apex of the lung and broncho-pneumonia does not. The diagnosis is substantiated by the history and course of the disease or by the presence of tubercle bacilli in the sputa.

(*b*) The extensive *atelectasis* of the lower lobes which may accompany broncho-pneumonia gives rise to dulness and absence of respiratory and vocal sounds. Thus, the signs of *pleuritic effusion* are simulated, and in children the possibility of empyema should not be forgotten. As a rule, broncho-pneumonia gives rise to much greater dyspnœa, and is associated with a more extensive bronchitis, than usually coexist with pleural effusion. The atelectatic lobules may be expanded by coughing or by the cutaneous stimulus of cold water, and thus resonance and breath sounds may suddenly return. With pleuritic effusions, of course, such a change is impossible.

(*c*) Empyema may be indistinguishable from broncho-pneumonia except by puncture or surgical operation (see above, p. 291).

2. *Chronic Broncho-Pneumonia*

Pulmonary induration was found in 12 out of 85 cases of broncho-pneumonia autopsied at the Massachusetts General Hospital (Lord). This corresponds with the clinical type of chronic broncho-pneumonia to which Riesman has recently called attention. It cannot be distinguished from the fibroid or chronic interstitial types of pneumonitis but is to be sharply separated from tuberculosis.

The physical signs are those of solidification with or without râles and—in extensive disease of long standing—evidence of contracted chest.

In many cases the disease complicates bronchiectasis or pulmonary abscess. It is commonest in children. From phthisis it is distinguished by its usually non-apical site, by its history and usually by the absence of fever, emaciation and constitutional symptoms. The sputum is alone decisive, however.

VI. Tuberculosis of the Lungs

1. *Incipient Tuberculosis*

In the earlier stages of the disease there may be absolutely no recognizable physical signs, and the diagnosis may be established only by the positive result of a *tuberculin* injection or by the combination of *debility, indigestion or loss of weight with slight fever* not otherwise to be accounted for.

In some cases the earliest evidence of the disease is *hæmoptyis.*[1] When a patient consults a physician on account of hæmoptysis, it is frequently impossible to find any physical signs of disease in the lungs; not until weeks or months later do the characteristic changes recognizable by physical examination make their appearance.

The very *early hoarseness* of the voice in tuberculous patients is of great importance and often attracts our attention to the lungs when the patient has said nothing about them. Definite physical signs in the lungs and tubercle bacilli in the sputa (artificially obtained through the use of potassic iodide, see below) may occasionally be demonstrated before any cough has appeared. On the other hand, the patient may cough for weeks before anything abnormal can be discovered in the lungs. Occasionally tuberculosis begins with an ordinarily bilateral bronchitis or broncho-pneumonia. I have found tubercle bacilli in four such cases. More often the earliest physical signs are:

(*a*) Fine crackling râles at the apex of one lung, heard only with or after cough and at the end of inspiration. (More rarely squeaks may be heard.)

(*b*) A slight diminution in the excursion of the diaphragm on the affected side, as shown by *x*-ray or by Litten's diaphragm shadow.

(*c*) Slight diminution in the intensity of the respiratory murmur, with or without a high pitched or interrupted inspiration ("*cog-wheel breathing*").

(*a*) In examining the apices of the lungs for evidence of early tuberculosis one should secure, if possible, perfect quiet in the room, and have the clothes entirely removed from the patient's chest. The ordinary hard-rubber chest-piece is better than the chest-piece of the Bowles instrument, and both the chest-piece and the skin should be wetted. After listening during quiet breathing over the apices above and below the clavicle in front, and above the spine of the scapula behind, the patient should be directed to breathe out and then, at the end of expiration, to cough. During this cough and the deep inspiration which is likely to precede or follow it, one should listen as carefully as possible at the apex of the lung, above and below the clavicle, concentrating attention especially upon the cough itself and upon the last quarter of the inspiration, when râles are most apt to appear. Sometimes only one or two crackles may be heard with each inspiration, and not infrequently they will not be heard at all unless the patient is made to cough, but even *a single râle, if persistent,*[1] is important. In children or others who cannot cough at will, one can accomplish nearly the same result by making them count as long as possible with one breath and then listening to the immediately succeeding inspiration. When listening over the apex of the lung, one should never allow the patient to turn his head sharply in the other direction, since such an attitude stretches the skin and muscles on the side on which we are listening so as to produce annoying muscle sounds or skin rubs. Lowered shoulders and a thoroughly relaxed attitude are essential.

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[1] Never percuss a patient within forty-eight hours after a hemorrhage, and never encourage cough or forced respiration in such a one. There is danger of starting a fresh hemorrhage.

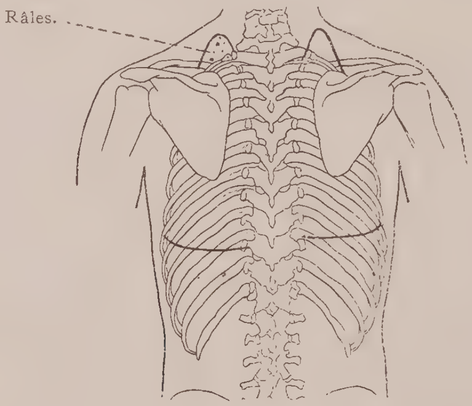

FIG. 176.—Diagram to Show Position of Earliest Signs in Tuberculosis.

In cases in which one suspects that incipient tuberculosis is present and yet in which no positive evidence can be found, it is a good plan to give iodide of potassium, (gr. vii, three times a day) for a few days. The effect of this drug is often to make râles more distinct, and sometimes to increase expectoration so that tubercle bacilli can be demonstrated when before none were to be obtained.[1]

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[1] Râles heard only during the first few breaths and not found to persist on subsequent examinations, may be due to the expansion of atelectatic lobules.

(*b*) The diminution in the excursion of the diaphragm upon the affected side in cases of incipient phthisis has been much insisted upon by F. H. Williams and others who have interested themselves in the radioscopy of the chest. Litten's diaphragm shadow gives us a method of observing the same phenomenon without the need of a fluoroscope. Even very slight tuberculous changes in the lung are sufficient to diminish its elasticity and so to restrict its excursion and that of the diaphragm. Comparisons must always be made with the sound side in such cases. It must be remembered that pleuritic adhesions, due to a previous inflammation of the pleura, may diminish or altogether abolish the excursion of the diaphragm shadow, independently of any active disease in the lung itself.

Some radiologists believe that they can detect the presence of tuberculosis in the lung by radioscopy at a period at which no other method of physical examination shows anything abnormal, but postmortem results rarely in my experience support this belief. In incipient tuberculosis the *x*-ray as often leads us wrong as right.

Interrupted or cog-wheel respiration, in which the inspiration comes in high-pitched irregular jerky puffs not synchronous with the cardiac impulse, signifies that the entrance of the air into the alveoli is impeded, and such impediment is most likely to be due to tuberculosis when present over a considerable period in a localized area of pulmonary tissue. It has, however, no relation to the activity of the process; indeed it may be heard in wholly arrested cases. Only *moisture*, crackles with cough, proves an active process.

2. *Moderately Advanced Cases*

So far I have been speaking of the detection of tuberculosis at a stage prior to the production of any considerable amount of solidification. The signs considered have been those of bronchitis localized at the apex of the lung, or of a slightly diminished pulmonary elasticity, whether due to pleuritic adhesions or to other causes. We have next to consider the signs in cases in which solidification is present, though relatively slight in amount. This condition is comparatively easy to recognize when it occurs at the left apex, but more difficult in case only the right apex is diseased. Partial solidification of a small area of lung tissue at the left apex gives rise to

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[1] Any irritating vapor—for example, creosote vapor—which produces violent cough and expectoration, may be used to expel bronchial secretions in doubtful cases. Tubercle bacilli may then be found in the sputum of patients who, without the irritating inhalation, have no cough and so no sputa, but this is in my opinion a dangerous procedure, as it may "light up" a quiescent process in the lung.

(*a*) Slight dulness on light percussion,[1] with increased resistance.

(*b*) Slight increase in the intensity of the spoken and whispered voice, and of the tactile fremitus (in many cases).

(*c*) Some one of the numerous varieties of broncho-vesicular breathing (true bronchial breathing is a late sign).

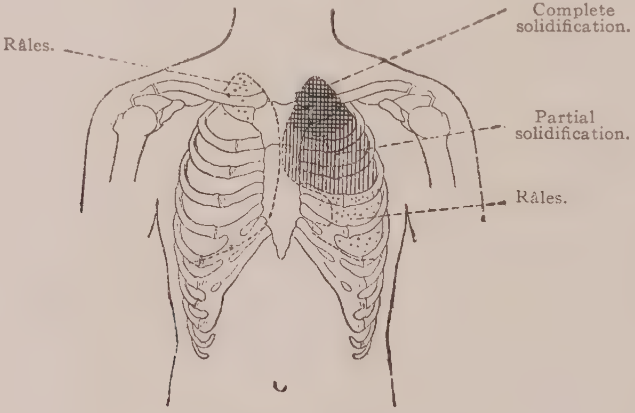

FIG. 177.—Diagram of Signs in Phthisis.

(*d*) Abnormally loud transmission of the heart sounds, especially under the clavicle.

In case there is also a certain amount of secretion in the bronchi of the affected area or ulceration around them, one often hears râles of a peculiar quality to which Skoda has given the name of "*consonating* râles." Râles produced in or very near a solidified area are apt to have a very sharp, crackling quality, their intensity being increased by the same acoustical conditions which increase the intensity of the voice sounds over the same area. When such râles are present at the apex of either lung, the diagnosis of tuberculosis is almost certain, but if, as not infrequently occurs, there are no râles to be heard over the suspected area, our diagnosis is clear *only* in case the signs occur at the left apex. Even these signs may mean only a healed tuberculosis, not an active process, unless râles can be elicited. Precisely the same signs, if present at the right apex, leave us in doubt regarding the diagnosis, for the reason that, as has been explained above, we find at the apex of the right lung, in health, signs almost exactly identical with those of a slight degree of solidification. Hence, if these signs, and only these, are discovered at the right apex, we cannot feel sure about the diagnosis until it is confirmed by the appearance of râles, the presence of fever, loss of weight, a positive tuberculin reaction (ocular, cutaneous or subcutaneous), by *x*-ray evidence or by the finding of tubercle bacilli in the sputum.[1]

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[1] Other causes of dulness, such as asymmetry of the chest, pleural thickening, and tumors, must be excluded. Emphysema of the lobules surrounding the tuberculous patch may completely mask the dulness.

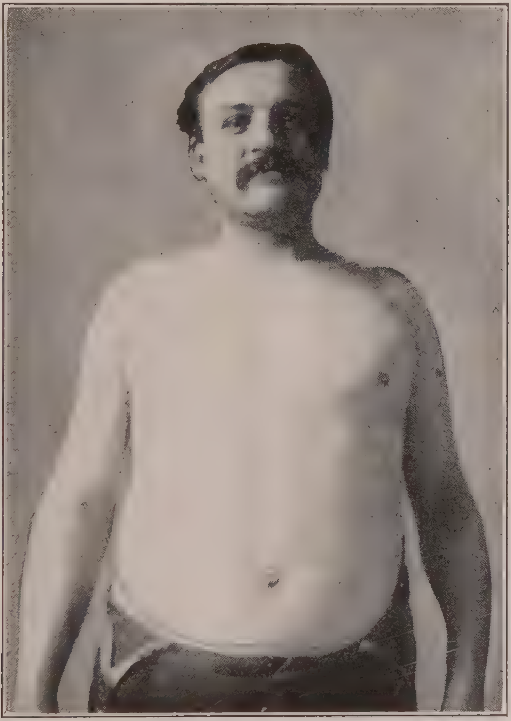

FIG. 178.—This Patient has Solidification at both Apices and Tubercle Bacilli in the Sputa. He feels perfectly well.

A sign characteristic of early tuberculous changes in the lung and one which I have frequently observed in the lower and relatively sounder lobes of tuberculous lungs is *a raising of the pitch of inspiration,*[1] without any other change in the quality of the breathing or any other physical signs. The importance of this sign in the diagnosis of early tuberculosis of the lungs was insisted upon by the elder Flint in his work on "The Respiratory Organs" (1866), and has more recently been mentioned by Norman Bridge. I have referred to this sort of breathing above as broncho-vesicular breathing of the first (*i.e.*, earliest) type (see p. 157).

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[1] The natural disparity between the two apices is less marked in the supraspinous fossa behind than over the clavicle in front, and hence pathological dulness at the apex is more often demonstrable behind than in front.

It must never be forgotten that tuberculosis may take root in the most finely formed chests and in persons apparently in blooming health. The "phthisical chest" and the sallow, emaciated figure of the classical descriptions apply only to very advanced cases. Fig. 178 represents a patient with moderately advanced signs of phthisis and abundant tubercle bacilli in the sputa. He feels perfectly well and is at work. On the other hand, a patient with very slight signs may be utterly prostrated by the toxæmia of the disease.

3. *Advanced Phthisis*

Characteristic of the more advanced stages of tuberculosis in the lungs is the existence of large areas of solidified and retracted lung, and, to a lesser extent, the signs of cavity formation. The patients are pale, emaciated, and feverish. The signs of solidification have already been enumerated in speaking of pneumonia. They are:

1. Marked dulness, or even flatness,[2] with increased sense of resistance.
2. Great increase of voice sounds or of tactile fremitus.
3. Tubular breathing, sometimes loud, sometimes feeble.
4. As a rule, coarse râles, due to breaking down of the caseous tissue, are also to be heard over the solidified areas. Sometimes these râles are produced within the pleuritic adhesions, which are almost invariably present in such cases. If they disappear just after profuse expectoration, one may infer that they are produced within the lung.

Increase in the intensity of the spoken voice, of the whispered voice, or of the tactile fremitus may be marked and yet no tubular breathing be audible. Each of these signs may exist and be of importance as signs of solidification without the others. As a rule, it is true, they are associated and form a very characteristic group, but there are many exceptions to this rule.

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[1] "Sharp breathing" (Turban).

[2] Unless senile emphysema masks it. Fibroid phthisis (*vide infra*) may show no dulness. Remember that gastric tympany may be transmitted to the left lung and mask dulness there.

In most advanced cases there is some *cardiac displacement* usually by traction of the adjacent lung and *usually to the left.*

Gerald Webb has called attention also to *displacement of the trachea* as felt at the suprasternal notch in many advanced or moderately advanced cases.

One of the common variations from the typical group of signs just mentioned occurs in the fibroid cases with pleural thickening. In these the breathing may be everywhere feeble, though the presence of a few râles with dulness and increased voice sounds makes the diagnosis obvious. In any almshouse group of old consumptives one finds (*a*) *cases in which one hears much* that strikes the novice—loud tubular breathing, abundant râles, etc., (*b*) *cases where there is very little to hear* with the stethoscope and that little not in itself distinctive. Some of these cases are probably of the peribronchial type recently discussed by Clive Riviere and others.

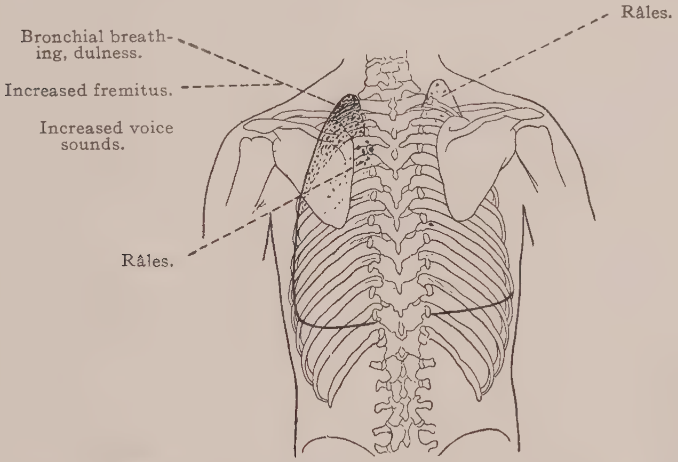

Fig. 179.—To Illustrate Progress of Signs in Pulmonary Tuberculosis.

The tendency of the spinal column to transmit to the sound lung sounds produced in an area of solidification immediately adjacent to it on the other side, has been already alluded to in the section on pneumonia, and what was then said holds good of tuberculous solidification. But since in tuberculosis both lungs are practically always involved *whether we find signs or not,* spinal transmission is not important.

Since solidification is usually accompanied by retraction in the affected lung in very advanced cases, the chest falls in to a greater or less extent over the affected area, and the respiratory excursion is much diminished, as shown by ordinary inspection and by the diminution or disappearance of the excursion of the diaphragm shadow. The intensity of the tubular breathing depends on the proximity of the solidified portions to the chest wall and to the large bronchi, as well as on the presence or absence of pleuritic thickening.

It is rare to find a whole lung solidified. *The process*, beginning at the apex or just below, *extends down as far as the fourth rib in front*, *i.e.*, through the upper lobe, *in a relatively short time, but below that point its progress is comparatively slow* and the lower lobes may be but little affected up to the time of death. On the relatively sound side, the exaggerated (compensatory) resonance may mask the dulness of a beginning solidification there, which sooner or later is sure to occur.

About the time that the tuberculous process appears in the previously sound lung it is apt to show itself at the apex of the lower lobe of the first affected. Consonating râles appear posteriorly along the line which the vertebral border of the scapula makes when the arm is raised over the shoulder. These points are illustrated in Fig. 179.

(*a*) *Cavity Formation*

Cavities of greater or lesser extent are formed in almost every case of advanced phthisis, but very seldom do they attain such size as to be recognizable during life. Indeed, the diagnosis of cavity in phthisis plays a much larger part in the text-books than it does in the practice of medicine, since to be recognizable by physical examination, a cavity must not only be of considerable size but its walls must be rigid and not subject to collapse,[1] it must communicate directly with the bronchus and be situated near the surface of the lung, and it must not be filled up with secretions. It can readily be appreciated that it is but seldom that all these conditions are present at once; even then the diagnosis of cavity is a difficult one, and I have often known skilled observers to be mistaken on this point.

The signs upon which most reliance is usually placed are:

(*a*) Amhporic or cavernous breathing.

(*b*) "Cracked-pot resonance" on percussion.

(*c*) Coarse, gurgling râles.

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[1] Yet not so rigid as to be uninfluenced by the entrance and exit of air.

(*a*) *Cavernous or Amphoric Respiration.*—When present, this type of breathing is almost pathognomonic of a cavity. It is also to be heard in pneumothorax, but the latter disease can usually be distinguished by the associated physical signs. Cavernous breathing differs from bronchial or tubular breathing in that its pitch is lower and its quality *hollow*. The pitch of expiration is even lower than that of inspiration. Since a pulmonary cavity is almost always surrounded by a layer of solidified lung tissue, we usually hear around the area occupied by the cavity a ring of bronchial breathing with which we can compare the quality of the cavernous sounds.

(*b*) Percussion sometimes enables us to demonstrate a circumscribed area of tympanitic resonance surrounded by marked dulness. More often the "cracked-pot" resonance can be elicited by percussing over the suspected area while the chest-piece of the stethoscope is held close to the patient's open mouth.

Cracked-pot resonance is often absent over cavities; rarely occurs in any other condition (*e.g.*, in percussing the chest of a healthy, *crying* baby, and occasionally over solidified lung).

(*c*) The voice sounds sometimes have a peculiar hollow quality (amphoric voice and whisper).

(*d*) Cough or the movements of respiration may bring out over the suspected area splashing or gurgling sounds, or occasionally a metallic tinkle. Flint has also observed a circumscribed bluging of an interspace during cough. Bruce noted a high-pitched sucking sound during the inspiration following a hard cough ("rubber-ball sound").

Very important in the diagnosis of cavity is the *intermittence of* all above-mentioned signs, which are present only when the cavity is comparatively empty, and disappear when it becomes wholly or mostly filled with secretions. For this reason, the signs are very apt to be absent in the early morning before the patient has expelled the accumulated secretions by coughing.

Wintrich noticed that the note obtained when percussing over a pulmonary cavity may change its pitch if the patient opens his mouth. Gerhardt observed that the note obtained over a pulmonary cavity changes if the patient shifts from an upright to a recumbent position. Neither of these points, however, is of any importance in diagnosis. The same is true of metamorphosing breathing (see above, p. 158).

Tuberculous cavities differ from those produced by pulmonary abscess or gangrene in that the latter are usually in the lower two-thirds of the lung. Bronchiectasis, if considerable in extent, cannot be distinguished by physical signs alone from a tuberculous cavity, but it is more often disseminated (like abscess or gangrene) in the lower part of the lungs especially.

4. *Hilus Tuberculosis* (*Peribronchial infiltration*)

We are indebted to *x*-ray work for the recent emergence of a type distinctly different from the ordinary apical tuberculosis and probably much rarer. Starting in the deep areas about the hilus where childhood infection occurs, a fresh active process may arise in adult life and spread outwards in any and all directions towards the surface. Though mentioned by occasional German writers since 1911, this form of tuberculosis has been practically unrecognized until in 1914–1918 it was popularized by American Army surgeons through the lessons learned by them of Col. Bushnell at Washington and in England by Clive Riviere.

Physical Signs

1. Retraction and dulness at *both* apices (narrowing of Krönig's isthmus).

2. In some cases an area of dulness between the spine of the scapula and the vertebral column—and corresponding to one or another lung root—can be percussed out. A similar area may be found in front.

3. The respiratory movements of the lung bases may be impaired or abolished.

4. Auscultation shows practically nothing.

5. *X*-ray shows increase of all the normal lung markings and an extension of these lines towards and to the surface of the lung. The more active and extensive the disease, the more mottling and shading of normally light areas around and between the bronchial lines.

All this might mean nothing but healed tuberculosis unless it is coupled with

6. *Constitutional symptoms* of active infection—fever, prostation, digestive disturbance, loss of weight, etc.—which are essential for diagnosis.

Later, râles may appear as the process reaches out to the lung surface, but this may never occur. In that case we have nothing but the percussion and *x*-ray signs of a healed hilus process *plus* constitutional signs of activity. Unless tubercle bacilli are to be found (and this is often impossible) there is no proof that the process is tuberculous. Statistical evidence, however, shows that such a process so situated is almost always tuberculous.

The process is often very slow and chronic in its course. Dyspnœa and chest pain are prominent.

From active apical phthisis this process differs by its scarcity or absence of râles, and its lack of unilateral predominance in the evidence of disease.

5. *Fibroid Phthisis*

This term applies to slow tuberculous or non-tuberculous processes with relatively little ulceration and much fibrous thickening especially about the bronchi.

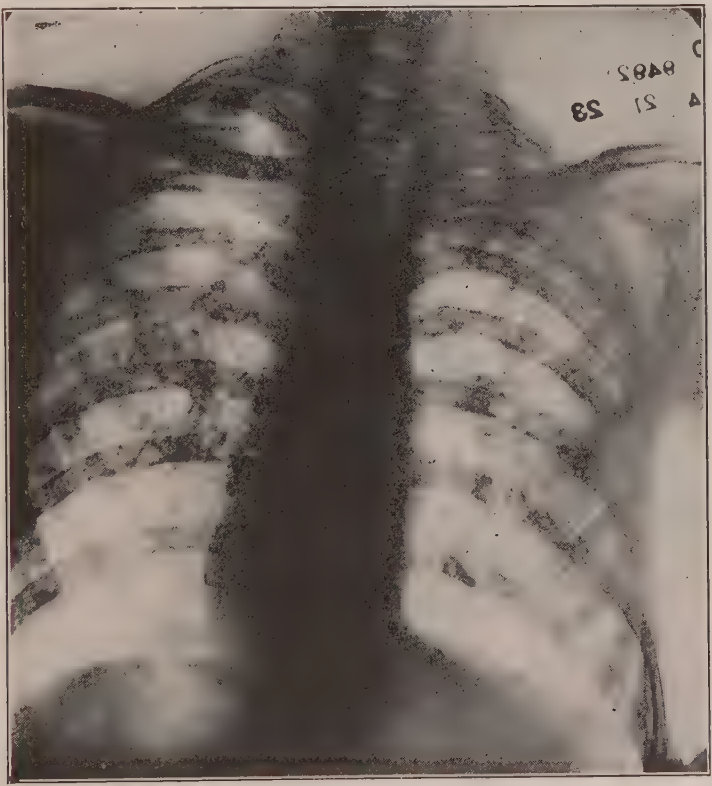

FIG. 180.—Pneumoconiosis with tuberculosis (right apex).

In a considerable number of cases the physical signs do not differ materially from those of the ordinary ulcerating forms of the disease, but occasionally when a slow chronic process at the apex of the lung results in the falling-away of the parenchyma of the lung so that we have left a cluster of bronchi matted together by fibrous tissue, the percussion note may be noticeably tympanitic; similar tympany may be due to emphysema of the lobules surrounding the diseased portion. In such cases râles are usually entirely absent; otherwise, the signs do not differ from those of ordinary phthisis, except that falling-in of the chest walls over the retracted lung may be more marked. Quite often the heart may be drawn toward the affected lobes, *e.g.*, upward and to the right in right-sided phthisis at the apex. In two cases of fibroid disease at the left base, Flint found the heart beating near the lower angle of the left scapula.

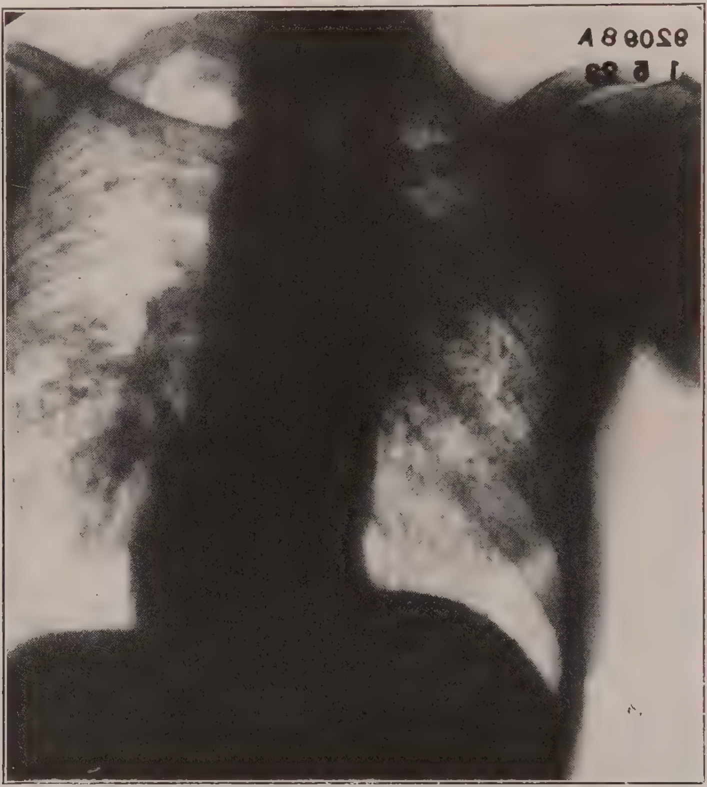

FIG. 181.—Pneumoconiosis.

6. *Phthisis with Predominant Pleural Thickening*

Tuberculosis in the lung is in certain cases overshadowed by the manifestations of the same disease in the pleura, so that the signs are chiefly those of *thickened pleura.* To this subject I shall return in the section on Diseases of the Pleura (see below, p. 343).

7. "*Emphysematous*" *Form of Phthisis*

Tubercle bacilli are not very infrequently found in the sputa of cases in which the history and physical signs point to chronic bronchitis with barrel chest. I have seen two cases within a year. Dulness is masked by hyperresonance, tubular breathing is absent, and piping and babbling râles are scattered throughout both lungs. The emphysema may be of the senile or small-lunged type, as in one of my recent cases (with autopsy), or it may be associated with huge downy lungs and the "barrel chest." Such cases cannot be identified as phthisis

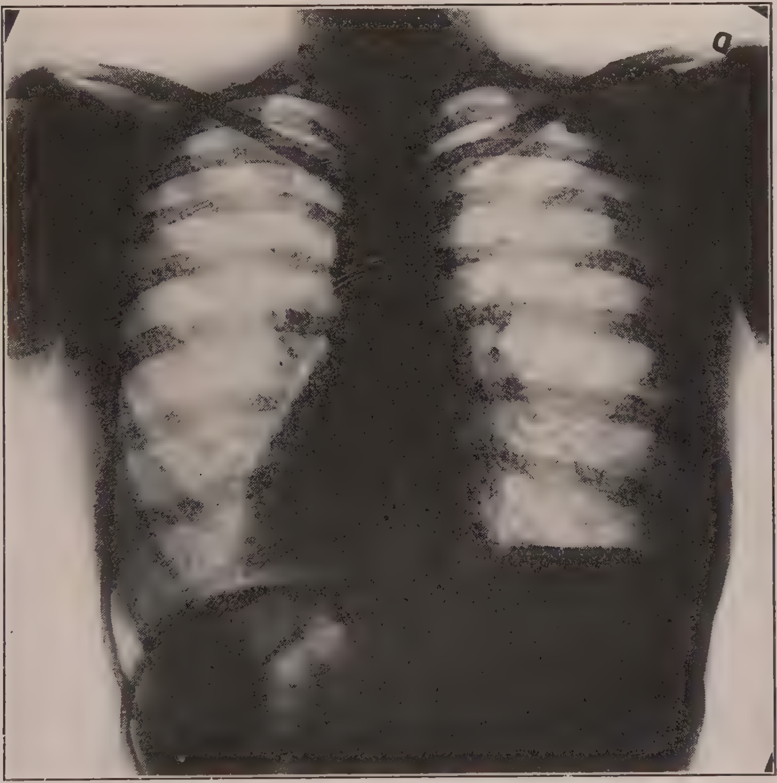

FIG. 182.—Miliary tuberculosis. Pleurisy at right base with adhesions to the diaphragm.

during life unless we make it an invariable rule to examine for tubercle bacilli the sputa of every case in which sputa can be obtained, *no matter what are the physical signs.*

8. *Phthisis with Anomalous Distribution of the Lesions*

I have never known tuberculosis to begin at the base of the lung. When the process seems to begin in this way, a healed focus is often to be found at one apex surrounded by a shell of healthy lung.

The summit of the axilla should always be carefully examined, as tuberculous foci may be so situated as to produce signs only at that point.

Another point often overlooked in physical examination is the *lingula pulmonalis* or tongue-like projection from the anterior margin of the left lung overlapping the heart. Tuberculosis is sometimes found further advanced at this point than anywhere else.

As a rule, cases in which signs like those of phthisis are found at the base of the lung turn out to be either empyema, bronchiectasis, abscess, or organized pneumonia (cirrhosis of the lung).

9. *Acute Pulmonary Tuberculosis*

No one of the three forms in which acute phthisis occurs, viz.,

(*a*) Acute tuberculous pneumonia,

(*b*) Acute tuberculous bronchitis and peribronchitis,

(*c*) Acute miliary tuberculosis, involving the lungs, can be recognized by physical examination of the chest. The first form is almost invariably mistaken for ordinary croupous pneumonia, until the examination of the sputa establishes the correct diagnosis. In the other two forms of the disease, the physical signs are simply those of general bronchitis.

CHAPTER XVIII

THE BARREL CHEST, ASTHMA, PULMONARY SYPHILIS, ETC.

I. "THE BARREL CHEST" AND ITS RELATION TO EMPHYSEMA

By pathologists cases of emphysema are divided into two groups.

(1) *Large-lunged* emphysema, usually associated with chronic cough and asthma.

(2) *Small-lunged*, or senile, emphysema.

In both conditions we have a dilatation and finally a breaking down of the alveolar walls until the air spaces are become relatively large and inelastic. In both forms, the elasticity of the lung is diminished; but in the large-lunged form we have an increase in the volume of the whole organ in addition to the changes just mentioned.

Whether there is any clinical picture, or any physical signs are recognizable as corresponding with these lesions I am quite uncertain. Till 1921 I thought I could recognize emphysema. The following facts have disillusioned me. In 12 cases diagnosed as emphysema at the Massachusetts General Hospital, only 3 showed any emphysema *post-mortem*. On the other hand, of the 153 cases demonstrated *post-mortem* only 7 were recognized in life. The following description refers to a clinical entity which we have been accustomed to call emphysema of the lungs. I am now convinced that this clinical entity (barrel chest, hyperresonance, feeble prolonged expiration etc.) is not always connected with emphysema and that the pathological entity "emphysema" is often present without recognisable physical signs. I know today nothing about the physical signs of emphysema. The thoracic condition about to be described is very possibly a disease or disorder of the chest walls themselves—leading to various changes in resonance, respiratory murmur, tactile fremitus, etc. As a guess, I will call this condition "The Barrel Chest" and describe under this provisional term what is usually described as emphysema. In typical cases the antero-posterior diameter of the chest is greatly increased, the interspaces are widened, and the costal angle is blunted, while the angle of Ludwig[1] becomes prominent. The shoulders are high and stooping and the neck is short (see Fig. 183). The patient is often considerably cyanosed, and his breathing rapid and difficult. Inspiration is short and harsh; expiration prolonged and difficult. The ribs move but little, and, owing to the ossification of their cartilages, are apt to rise and fall as if made in one piece (*en cuirasse*). The working of the auxillary muscles of respiration is not infrequently seen. The diaphragm shadow (Litten's sign) begins its excursion one or two ribs farther down than usual and moves a much shorter distance than in normal cases.

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[1] Formed by the junction of the manubrium with the second piece of the sternum.

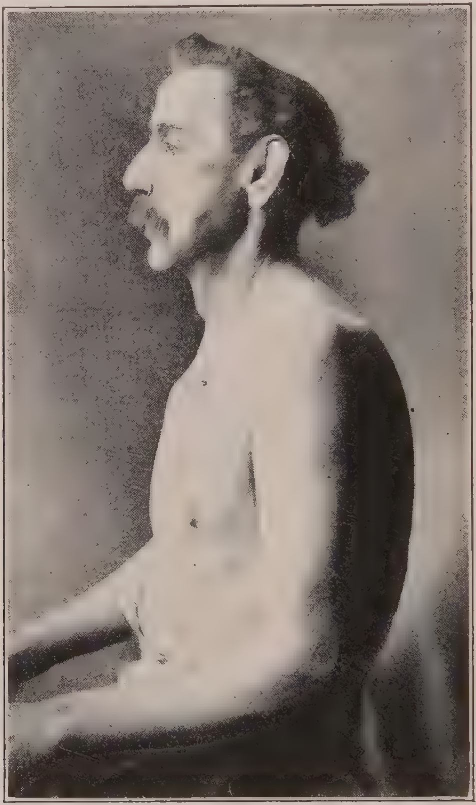

FIG. 183.—Barrel Chest with Chronic Bronchitis.

Palpation shows a diminution in the tactile fremitus, throughout the affected portions; that is, usually throughout the whole of both lungs. Sometimes it is scarcely to be perceived at all.

Percussion yields very interesting information. The disease manifests itself—

(*a*) By hyperresonance on percussion, with a shade of tympanitic quality in the note.

(*b*) By the extension of the margins of the lung so that they encroach upon portions of the chest not ordinarily resonant.

The degree of hyper-resonance depends considerably upon the thickness of the chest walls. The note is most resonant and has most of the tympanitic quality when the disease occurs in old persons with relatively thin chest walls. The encroachment of the hyperresonant areas upon the liver and heart is demonstrated by the lowering of the line of liver flatness from its ordinary position at the sixth rib to a point one or two interspaces farther down or even to the costal margin, while the area of cardiac dulness may be altogether obliterated. At the apices of the lungs resonance may be obtained one or two centimetres higher than normally and the quality may be markedly tympanitic. In the axillæ and in the back the pulmonary resonance extends down one inch more below its normal position.

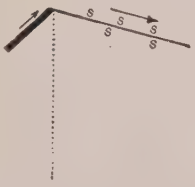

FIG. 184.—Diagram to Illustrate "Barrel Chested" Breathing with Musical Expiratory Râles.

Auscultation shows in uncomplicated cases no very marked modification of the inspiratory murmur, which, however, may be shortened and enfeebled. The most striking change is a *great prolongation and enfeeblement of expiration*, with a lowering of its pitch (see Fig. 184). Owing to an accompanying bronchitis or asthma, squeaky râles are often heard during expiration, but their high pitch is to be distinguished from the low pitch of the accompanying respiratory murmur.

This type of breathing is like bronchial breathing in one respect; namely, that in both of them expiration is made prolonged, but "barrel chested" breathing is feeble and low-pitched, while bronchial breathing is intense and high-pitched. At the bases of the lungs the respiration is especially feeble and may be altogether replaced by cracking râles.

(*a*) *Summary*

1. Hyperresonance on percussion.
2. Feeble breathing with prolonged expiration.
3. Diminished fremitus and voice sounds.
4. Encroachment of the resonant areas on the heart and liver dulness.

(b) *Differential Diagnosis*

(*a*) The barrel chest and its results may be confounded with *pneumothorax*, since in both conditions hyperresonance and feeble breathing are present. But the former is usually bilateral, does not displace neighboring organs, and is not often associated with hydrothorax. The barrel chest is usually associated with cardiac weakness and so with squeaking or bubbling râles, while in pneumothorax breathing is absent or distant amphoric without râles.

(*b*) The signs of *aneurism* of the aorta pressing on the trachea or on a primary bronchus are sometimes overlooked because the foreground of the clinical picture is occupied by the signs of a coexisting barrel chest with bronchitis. The cough and wheezing which the presence of the aneurism produces may then be accounted for as part of the long-standing bronchitis, and the dulness and thrill over the upper sternum to which the aneurism naturally gives rise may be masked by extension of lung borders. But the evidence of pressure on mediastinal nerves and vessels (aphonia, unequal pulse or pupils, etc.), and the presence of a diastolic shock and tracheal tug are usually demonstrable; the danger is that we shall forget to look for them.

(*c*) The occurrence of a barrel chested form of *phthisis* I have already mentioned in discussing the latter disease (see p. 307).

2. *Barrel chest with Arterio-sclerosis and Asthma*

In the great majority of cases, the barrel chest is associated with weak heart, pulmonary edema, and asthmatic paroxysms. Such association is especially frequent in elderly men who have had a winter cough for many years and in whom arterio-sclerosis is more or less well marked. In such cases the prolonged and feeble expiration is usually accompanied by squeaking and groaning sounds, or by moist râles of various sizes and in various parts of the chest. When the asthmatic element predominates, dry râles are more noticeable, and occur chiefly or wholly during expiration, while inspiration is reduced to a short, quick gasp.

3. *Interstitial Emphysema*

In rare cases, violent paroxysms of coughing may rupture the walls of the alveoli so as to allow the passage of air into the interstitial tissue of the lung, whence it may work through so as to manifest itself under the skin, giving rise to a peculiar crackling sensation on palpation, and to a similar sound on auscultation. In the influenzal pneumonias of 1918 this complication was often seen. The air appeared first under the skin of the neck whence it worked down over the chest and sometimes over the entire body and face. It is later absorbed and does not increase the gravity of the prognosis.

4. "*Complementary Emphysema*"

When extra work is thrown upon one lung by loss of the function of the other, as in pleuritic effusion—a considerable stretching of the overworked sound lung may take place. The elasticity of the lung is not diminished as in emphysema, but is greatly increased. Hence the term complementary emphysema should be dropped and the term complementary (or compensatory) *hyperresonance* substituted.

Like the barrel chest, this condition leads to encroachment of the hyperresonant areas upon the neighboring organs (as shown by a reduction in the area of dulness corresponding to them), but the respiratory murmur is *exaggerated* and has none of the characteristics of barrel chested breathing.

A word may here be added regarding the condition described by West under the name of

5. *Acute Pulmonary Tympanites*

In fevers and other acute debilitating conditions West has observed that the lungs may become hyperresonant and somewhat tympanitic on percussion, owing, he believes, to a loss of pulmonary elasticity. The tympanitic note, often observable around the solidified tissue in pneumonia, is to be accounted for, he believes, in the same way. Like the shortening of the first heart sound, acute pulmonary tympanites points to the weakening of muscle fibre which toxæmia is so apt to produce. Apparently the muscle fibres of the lung suffer like those of the heart.

II. BRONCHIAL ASTHMA

1. *Primary Spasm of the Bronchi*

During a paroxysm of bronchial asthma our attention is attracted even at a distance by the loud, weezing, prolonged expiration preceded by an abortive gasping inspiration. The breathing is labored, much quickened in rate, and cyanosis is very marked. The chest is distended and hyper-resonant, the position of the diaphragm low and its excursion much limited, and the cardiac and hepatic dulness obliterated by the resonance of the distended lungs. On auscultation, practically no respiratory murmur is to be heard despite the violent plunging of the chest walls. We hear squeaks, groans, muscular rumbles, and a variety of strange sounds, but amid them all practically nothing is to be heard of the *breath sounds*. "The asthmatic storm flits about the chest, now here, now there," the râles appearing and disappearing.

At the extreme base of the lungs there may be dulness due to atelectasis of the thin pulmonary margins.

(*a*) *Differential Diagnosis*

True broncho-spasm due to abnormal sensibility to pollens, horse or cat hair, food proteins, bacterial proteins and other irritants is to be distinguished from

(*a*) Mechanical irritation of the bronchi, by broncho-stenosis, aneurism or enlarged glands, may set up a spasm of the neighboring bronchioles much resembling that of primary bronchial asthma, but thorough examination should reveal other evidence of mediastinal pressure, and the history of the case is very different from that of asthma.

(*b*) *Spasm of the glottis* produces a noisy dyspnœa, but the difficulty is with *inspiration*, instead of with expiration, and the crowing or barking sound is not like the long wheeze of asthma. No râles are to be heard, and the signs in the lungs are those of collapse instead of the distention characteristic of asthma.

(*c*) The paroxysmal attacks of dyspnœa, which often occur in chronic nephritis, chronic bronchitis, arterio-sclerosis, and other diseases of the heart and kidney, may be entirely indistinguishable from primary bronchial asthma but for the evidence of the underlying cardiac of renal disease.

(*d*) Acute dyspnœa in young infants is sometimes due to enlarged thymus (*thymic asthma*). The diagnosis rests on the elimination of all other causes for sudden dyspnœa and the presence (sometimes) of increased substernal dulness when the child is put face downward.

III. Syphilis of the Lung

The diagnosis cannot be made with certainty from the physical signs, and rests entirely (in the rare cases in which it is made at all) on the history, the evidence of syphilis elsewhere in the body, and the result of treatment. Most cases are mistaken for phthisis.

Any case supposed to be phthisis, but in which the examination of the sputa for tubercle bacilli is repeatedly negative, should be given a course of anti-syphilitic treatment.

The physical signs, as in phthisis, are those of localized bronchitis or of solidification, but *the lesions are apt to be extensive in one lung and absent in the other.* Cavities are not formed.

1. *Bronchostenosis*

Aside from foreign bodies, the usual cause of stenosis in the bronchi is a syphilitic cicatrix at or near the tracheal bifurcation.

Occasionally an aneurism or a neoplasm may exert narrowing and irritative pressure from within or from without.

The physical signs are: (*a*) Dyspnœa. (*b*) Stridor or whistling sounds over the affected bronchus. (*c*) Diminution of resonance, breath sounds, and voice over the corresponding lung.

Syphilitic structures sometimes bleed. Foreign bodies, strange to say, may get into a bronchus and lie there forgotten for months or years till discovered by *x*-ray in the course of a search for the cause of a chronic couch, a bronchiectasis, lung abscess, or chronic broncho-pneumonia.

IV. BRONCHIECTASIS (BRONCHIAL DILATATION)

(*a*) The commonest type is that associated with chronic pneumonitis and recurrent attacks of winter cough. Innumerable small bronchioles become dilated and the resulting cavities are repeatedly infected—usually with influenza bacilli (Wm. H. Smith). The signs may be simply those of a chronic bronchitis with or without emphysema and asthmatic seizures, but the appearance of profuse *purulent* (not muco-purulent) sputa in rounded masses is distinctive. Foci of broncho-pneumonia appear from time to time with acute febrile attacks. In summer, the cavities are often dry and uninfected.

(*b*) When the disease is further advanced and the cavities are larger, a sudden change in the patient's position (or especially hanging head downward over the side of the bed) may cause him to raise large amounts of sputa (half a pint or more) within a few minutes. This sputum is not usually foul and rarely contains blood or elastic fibres. Even at this stage there may be no physical signs of localized cavities—but only those of the associated bronchitis, usually more marked at one base than at the other and associated with signs of partial solidification.

(*c*) In a small number of cases signs of cavity (see above p. 302) may be made out. As a rule the disease is widespread, but 44 per cent. of cases are confined to one lung, and 25 per cent. to one lobe. In about one-third of the cases there is a chronic fibrous type of tuberculosis in the background. Emphysema accompanies one-fourth of all cases and there were pleural adhesions in 32 of Lord's 42 cases. Hæmoptysis occurs in nearly one-half.

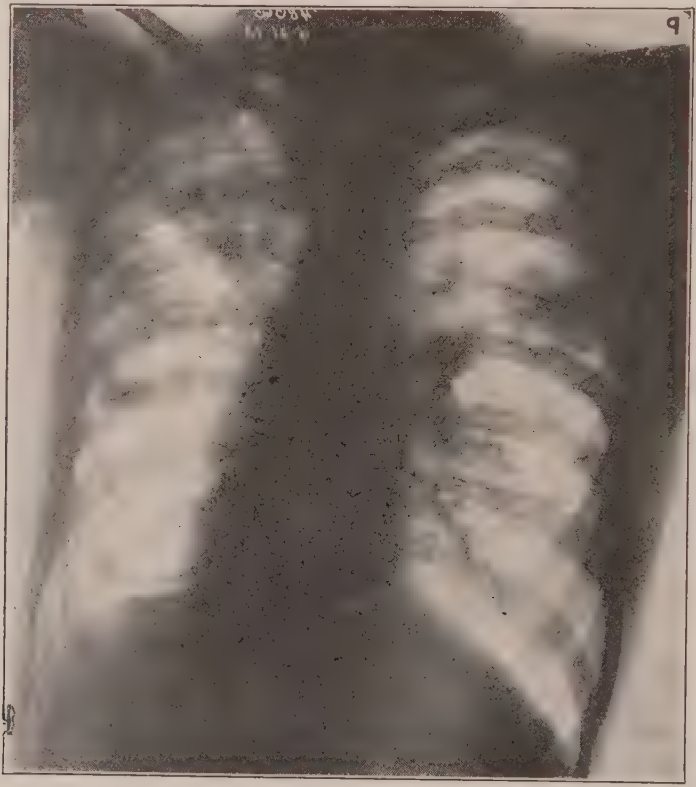

FIG. 185.—Bronchiectasis (bilateral).

Bronchiectasis is distinguished from abscess of the lung because the latter shows elastic tissue in the sputa, more often foul; evidence of general infection and of local cavity is oftener obtained in abscess. The *x*-ray helps little when the usual causes of abscess (foreign bodies) are absent, but one must remember that a foreign body or a cancerous nodule blocking a large bronchus may produce bronchiectasis. In such cases *x*-ray is our main reliance.

The disease may cause marked retraction of the chest on the affected side, and neighboring organs may be drawn out of place.

V. EXAMINATION OF SPUTA

1. *Origin*

Probably the majority of all sputa, excepting tobacco juice, come from the *nasopharynx*, and are *hawked*, not coughed up. It is rarely of value to examine such sputa, although influenza bacilli, diphtheria bacilli, pneumococci, and other bacteria may be found.

What we want in most cases is sputa coughed up from the primary bronchi or lower down, and the patient should be accordingly instructed. Early morning cough is more likely to bring up sputa from the bronchi.

Young children do not raise sputum, but when it is important to obtain it we may insert the forefinger (covered with a bit of cotton) into the pharynx, so as to excite a spasm of coughing. The sputumi is deposited on the cotton before the child has time to swallow it, and may then be withdrawn and examined.

2. *Quantity*

If the amount expectorated is large (*i.e.*, one-half a pint or more in twenty-four hours), we may be dealing with:

1. Pulmonary œdema (watery, sometimes pink and frothy).
2. Advanced phthisis (muco-purulent).
3. Empyema ruptured into a bronchus (pure pus not separated into pellets).
4. Abscess of the lung (foul smelling). Foul odor also occurs rarely in
5. Bronchiectasis (large amount of pure pus in pellets often expectorated within a few minutes on change of position).

3. *Odor*

Unless retained in a lung cavity (abscess, gangrene) sputum is rarely ill-smelling. In gangrene of the lung the breath as well as the sputum is horribly offensive, and the odor soon fills the room and the house.

4. *Gross Appearances*

(*a*) *Bloody sputum* (hæmoptysis) means *pure or nearly pure blood in considerable quantity*, a teaspoonful or more, not mere streaks of blood in muco-purulent sputum, which comes from an irritated throat and is of no importance.

Hæmoptysis thus defined is seen chiefly in the following conditions, arranged in the order of frequency:

1. Phthisis.
2. Pulmonary congestion with infarction (mitral disease), typhoid fever and other causes of peripheral thrombi.
3. Pneumonia.
4. After epistaxis.
5. Abscess of the lung or bronchiectasis.
6. Cancer of the lung.
7. Without known cause ("vicarious menstruation," etc.).

Rare causes are parasites (Distomum Westermanni), aortic aneurism rupturing into an air tube, ulcer of the trachea or bronchi (usually syphilitic).

The cause of hæmoptysis can usually be made out by a thorough examination of the chest and a study of the other symptoms in the case. *In phthisis there are often no physical signs in the lungs at the period when the bleeding occurs or for some weeks after it.* Blood *coughed up* can usually be distinguished from *blood vomited* (*hæmatemesis*) by careful questioning and by examining the blood. Blood coughed up often contains bubbles of air and is alkaline in reaction, while blood from the stomach is usually mixed with food, not frothy, and perhaps acid in reaction.

(*b*) *Pneumonic Sputum.*—The color is most characteristic; it is either

(1) Tawny-yellow or fawn-colored ("rusty"), or

(2) Orange-juice colored (*not* orange, but pale straw-colored)

These colors, associated with *great tenacity*, so that the sputum clings to the lips and does not fall from an inverted sputum-cup, are almost pathognomonic of pneumonia—though pneumonia often occurs without any such sputa.

(*c*) *Serous sputum*, profuse and watery, is characteristic of pulmonary œdema.

(*d*) *Black or gray sputum* is due to carbon, dust, or tobacco smoke inhaled.

(*e*) *Pure pus*—not muco-purulent—is oftenest seen in bronchiectasis, occasionally in empyema breaking through the lung.

(*f*) *Muco-purulent* sputum occurs in many diseases and is characteristic of none.

5. *Microscopic Examination*

Ninety-nine-one-hundredths of all examinations are for the tubercle bacillus. Of the many useful methods of staining for this organism the following seems to me the best:

1. Pick out with forceps the most purulent portion of the sputa and smear it *thinly* over a cover glass. All particles thick enough to be opaque should be removed from the cover glass before staining.

2. Dry the preparation *held in the fingers* over a Bunsen or alcohol flame. Then fix it in Cornet's forceps and pass it three times through the flame, sputum side down.

3. Flood it with carbolic fuchsin,[1] and steam it—do *not* boil it—over the flame for about thirty seconds. Be sure to use enough stain so that it does not dry on the cover glass.

4. Wash in water and decolorize for twenty seconds in twenty-per-cent. H_2SO_4.

5. Wash in water and then in ninety-five-per-cent. alcohol for thirty seconds or until the color ceases to come out.

6. Wash in water and cover with Löffler's methylene blue[2] for about thirty seconds.

7. Wash in water, dry on bottling paper, and mount in Canada balsam.

The whole process need not take more than five minutes, and every physician should be familiar with it.

The bacilli are stained red, everything else blue. They should be looked for only with an immersion lens (one-twelfth-inch), a wide-open diaphragm, and a good white light. In the vast majority of cases the bacilli are found, if at all, within a few minutes and in almost every field. Occasionally one has to search longer, but it is better to search one well-stained preparation thoroughly than to spend the time in preparing and examining several.

The *presence of red-stained bacilli* in specimens of sputa so prepared is practically pathognomonic of tuberculosis. Other acid-resisting bacilli occur in the urine, but almost never in the lung.

The *absence of tubercle bacilli* after at least six examinations of satisfactory specimens[3] obtained several days apart makes it very unlikely that phthisis is present. One or two negative examinations are of no significance.

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[1] Carbolic-acid crystals, 5 gm.; fuchsin (saturated alcoholic solution), 10 gm.; water, 100 gm.

[2] Saturated alcoholic solution of methylene blue, 30 c.c.; aqueous solution of KOH (1 in 10,000), 100 c.c.

[3] A satisfactory specimen is one prepared without any slips in technique from purulent sputa obtained by coughing and not by hawking.

Pneumococcic and Influenza Bacilli.—For both these organisms *Gram's stain* is on the whole the best. This is performed as follows:

1. Prepare a smear as above directed.
2. Cover it with aniline-oil-gentian-violet solution[1] (freshly made each week) and heat to steaming point.
3. Wash in water and cover with IKI solution[2] for thirty seconds.
4. Wash in ninety-five-per-cent. alcohol until the blue color ceases to come out.

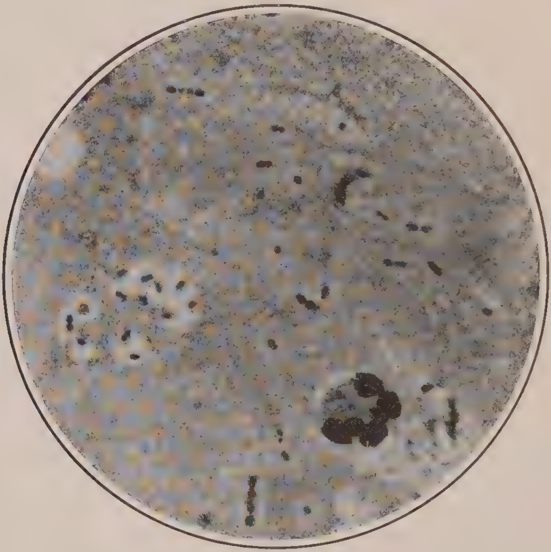

FIG. 186.—Pneumococci in Sputum. (W. H. Smith.) (Gram's stain.)

5. Counterstain with Bismarck, brown for thirty seconds.
6. Wash in water and mount in Canada balsam.

The pneumococcus with this stain comes out blue-black and its morphology is well shown (see Fig. 186). The presence of a few pneumococci free in the sputum is not of importance. When the organisms are very abundant, and especially when many of them are contained within leucocytes, a pneumococcus infection is strongly suggested, though it may be a pneumococcus bronchitis without pneumonia. In the earlier stages of an infection, fewer organisms are found within leucocytes than is the case later. Obviously one can learn only by practice what is meant by "few" or "many" organisms.

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[1] Saturated alcoholic solution of gentian violet, 13 c.c.; aniline water, 84 c.c. aniline water is the *clear* filtrate from the mixture of aniline, 5 parts, with water, 25 parts.

[2] Iodine, 1 gm.; potassium iodide, 2 gm.; water, 300 c.c.

The *influenza bacillus* is the smallest organism to be found in the sputum. In specimens stained by Gram's method (as above given) the influenza bacilli come out as minute, faintly brown-stained points, contrasting with the intense blue-black of pneumococcus and other organisms. Only when present in large numbers both inside and outside the leucocytes of the sputa are they diagnostic of actual influenzal infection, since the organism is a common inhabitant of the upper air passages.

Although other organisms—staphylococcus, micrococcus catarrhalis, streptococcus, bacillus mucosus capsulatus—are sometimes found in sputa, their importance does not justify an account of them here.

Indications for Sputum Examination.—Any cough with sputa lasting more than a week calls for an examination of sputa. In doubtful cases of influenza or pneumonia, and in any case in which tuberculosis is suspected an examination is imperative.

When the symptoms or physical signs suggest tuberculosis, but no sputa can be obtained, it is well to stimulate the bronchial secretions with 10 gr. of potassium iodide after meals for a week. A way of getting sputa from young children has already been described (page 303).

CHAPTER XIX

DISEASES AFFECTING THE PLEURAL CAVITY

I. HYDROTHORAX (PLEURAL DROPSY)

In cases of nephritis or of cardiac weakness due to vascular or cardiac disease a considerable accumulation of serum may take place in both pleural cavities. The physical signs are identical with those of pleuritic effusion (see below, page 330) except that the latter is almost always unilateral, while hydrothorax is usually bilateral. Exceptions to this rule occur, however, *especially on the right side* or in cases in which one pleural cavity has been obliterated by fibrous adhesions, the results of an earlier pleurisy.[1] The fluid obtained by tapping, in cases of hydrothorax, is usually considerably lower in specific gravity and poorer in albumin than that exuded in pleuritic inflammation.

The fluid shifts more readily with change of position than is the case with many pleuritic effusions, owing to the absence of adhesions in hydrothorax. But shifting of fluid can never be relied upon in the diagnosis either of hydrothorax or of pleural exudate.

Friction sounds, of course, do not occur, as the pleural surfaces are not inflamed.

II. PNEUMOTHORAX

Pneumothorax, or the presence of air in the pleural cavity, may result from wounds of the chest wall, but in 78 per cent. of cases it is a complication of tuberculosis which weakens the lung until by a slight cough or even by the movements of ordinary respiration the pulmonary pleura is ruptured and air from within the lung leaks into the pleural cavity. Biach (quoted by Fussell in Monographic Medicine, Vol. V, p. 388) found in 914 cases of pneumothorax the following causes:

Pulmonary tuberculosis	715 cases (78%)		92.7%
Pulmonary gangrene or abscess	75 cases	134 cases (14.6%)	
Empyema	45 cases		
Bronchiectasis	10 cases		
Pulmonary infarct	4 cases		
Trauma	35 cases	29 or 30%	
Other known causes	15 cases		
Unknown cause	14 cases		

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[1] In about two-thirds of all cases hydrothorax is either confined to the right side or more abundant in the right than in the left pleura. Only in about 17 per cent. of cases is it confined to the left side.

Hence disease of the lung or wounds cause 97 per cent. of the cases of pneumothorax.

If the opening is of considerable size, and the air is not hindered or encapsulated by adhesions, great and sudden dyspnœa with pain and profound "shock" may result. But in about two-thirds of all cases the onset is insidious, the air enters the pleural cavity gradually, the other lung has time to hypertrophy, and the heart and other organs become gradually accustomed to their new situations.

1. *Physical Signs*

1. *Inspection.*—The affected side may lag behind considerably in the movements of respiration. In very marked cases it is almost motionless and the interspaces are more or less obliterated. The diaphragm is much depressed and Litten's sign absent. In right-sided pneumothorax, which is relatively rare, the liver is depressed and its edge can be felt below the ribs.

The heart is displaced as by pleuritic effusion, but usually to a less extent. With left-sided pneumothorax the cardiac impulse may be lowered as well as displaced, owing to the descent of the diaphragm.

2. *Palpation.*—Fremitus is usually diminished or absent over the lower portions of the chest corresponding to the effused air. At the summit of the chest over the retracted lung, fremitus is also diminished or absent, as a rule (W. B. James). In rare cases when the lung is adherent to the chest wall and cannot retract, fremitus is preserved.

The positions of the heart and liver are among the most important points determined by palpation. Not infrequently no cardiac impulse is to be obtained. Sometimes it may be felt to the right of the sternum (see Fig. 188) or in the left axilla, but not infrequently it is so fixed by pleuropericardial adhesions that it is drawn upward toward the retracted lung or remains near its normal situation. The liver is greatly depressed in cases of right sided pneumothorax, and may be felt as low as the navel.

3. *Percussion.*—Loud tympanitic resonance is the rule throughout the affected side. Even a small amount of air is sufficient to render the whole side tympanitic and often to obscure the dulness which the frequently associated pleural effusion would naturally produce. Indeed, it is the rule that small effusions are wholly masked by the adjacent tympany.

In no other disease do we get such clear, intense tympanitic resonance over the chest.

The only exception to this rule occurs in cases in which the air within the chest is under great tension, making the chest walls so taut that, like an over-stretched drum, they cannot vibrate properly. Under these conditions the percussion note becomes muffled, at times almost dull.

Areas of dulness corresponding to the displaced organs (heart or liver) may sometimes be percussed out.

4. *Auscultation.*—Respiration and voice sounds are usually inaudible in the lower portions of the chest. At the top of the chest, and rarely in the lower parts, a faint amphoric or metallic breathing may be heard, but as a rule the amphoric quality is brought out much better by cough which is followed by a ringing after-echo. In W. B. James's[2] ninety cases the breathing was amphoric in thirty-one, diminished or absent in fifty-three, bronchial in six. Or the air in the pleura may be set to vibrating and made to give forth its characteristic, hollow, ringing sound if a piece of metal (*e.g.*, a coin) be placed on the back of the chest and struck with another coin, while we listen with the stethoscope over the front of the chest opposite the point where the coin is. In the combined statistics of Emerson[1] and James[2] the signs were present in forty-five out of sixty cases, or seventy-five percent.

The clear ringing sound heard in this way is quite different from the dull chink obtainable over sound lung tissue.

The "falling-drop sound" or "metallic tinkle," (see above, p. 170), was heard in thirty out of thirty-five of James's cases.

On the sound side the breath sounds are exaggerated. At the top of the affected side over the collapsed lung the breathing is bronchial and râles are occasionally heard.

In about 80 per cent. of cases pneumothorax is complicated by an effusion of fluid in the affected pleural cavity and we have then the signs of

III. Pneumohydrothorax or Pneumopyothorax

When both fluid and air are contained in the pleural cavity, the patient may himself be able to hear the splashing sounds which the movements of his own body produce. These are more readily appreciated if the observer puts his ear against the patient's chest and then shakes him briskly. Splashing sounds heard within the chest are absolutely pathognomonic and point only to the combination of fluid and air within the pleural cavity. One must distinguish them, however, from similar sounds produced in the stomach. By observing the position of maximum intensity of the sounds, this distinction may be easily made. Unfortunately the critical condition of the patient may make it impossible to try succussion, as in the acute cases with great shock it is dangerous to move patients at all.

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[1] Emerson: Pneumothorax, Johns Hopkins Hospital Reports, 1903, Vol. XI.

[2] Osler's Modern Medicine, Vol. III, p. 881.

The movements of breathing or coughing may bring out a "metallic tinkle" (see above, p. 170). At the base of the chest, over an area corresponding to the position of the fluid, an area of dulness may be marked out by percussion, and this area *shifts markedly* with change of position. The shifting dulness of pneumohydrothorax is strongly in contrast with the difficulty of obtaining any such shift in ordinary pleuritic effusion or in hydrothorax without air.

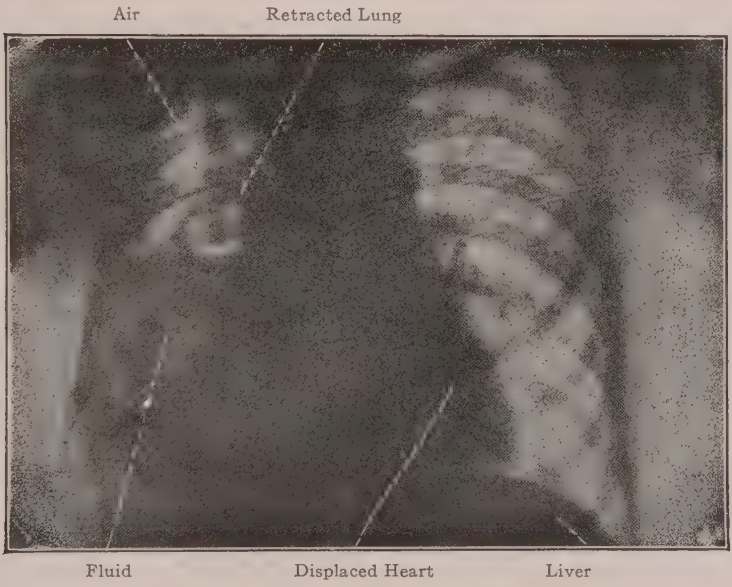

FIG. 187.—Left Pneumohydrothorax Seen from Behind. Note the horizontal line at the surface of the fluid and the retracted lung just above the inner half of this line. The heart is displaced to the right. Compare Fig. 188. (From v. Ziemssen's Atlas.)

(The distinction between "*open pneumothorax*," in which the rent in the lung through which the air escaped in the pleura remains open, and "*closed pneumothorax*," in which the rent has become obliterated—is one which cannot be established by physical signs alone. It is often said that amphoric breathing, and especially an amphoric ring to the voice and cough sounds, denote an open pneumothorax, but post-mortem evidence does not bear this out. Practically, an open pneumothorax is one in which the amount of effused air increases and closed pneumothorax is one in which the physical signs remain stationary.)

1. *Differential Diagnosis*

The distinction between pneumothorax and emphysema has already been discussed.

(*a*) When the air in the pleural sac is under such tension that the percussion note is dull, the physical signs may simulate pleuritic effusion, but real flatness, such as characterizes effusion, has not, so far as I know, been recorded in pneumothorax, and the sense of resistance on percussing is much greater over fluid than over air. In case of doubt puncture is decisive.

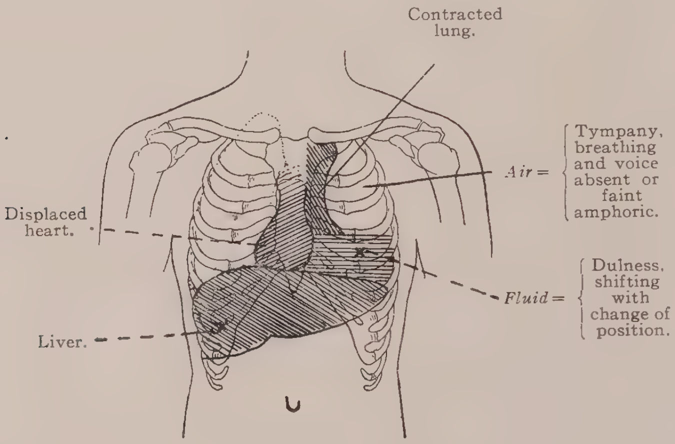
Contracted lung.
Tympany, breathing and voice absent or faint amphoric.
Air =
Displaced heart.
Dulness, shifting with change of position.
Fluid =
Liver.

FIG. 188.—Left Pneumoserothorax with Displaced Heart.

(*b*) Acute pneumothorax, coming on as it does with symptoms of collapse and great shock may be mistaken for angina pectoris, cardiac failure, embolism of the pulmonary artery, or acute pulmonary tympanites (see above, p. 313).

From all these it can be distinguished by the presence of a displaced heart and of amphoric or metallic sounds which are never to be obtained in the other affections named.

(*c*) Hernia of the intestine through the diaphragm (see Fig. 189) due to great weakening of the diaphragmatic muscular fibres, allows the intestines to encroach upon the thoracic cavity and simulate pneumothorax very closely. The history and course of the case, the abdominal pain, and vomiting, generally suffice to distinguish the condition. The peristalsis of the intestine may go on even in the thorax, and gurgling metallic sounds corresponding to it and unlike anything produced in the thorax itself may be audible.

The distinction between open and closed pneumothorax, to which I have already alluded, is far less important than the presence or absence of

(*a*) Pulmonary tuberculosis.

(*b*) Encapsulating adhesions in which the air is confined to a circumscribed area.

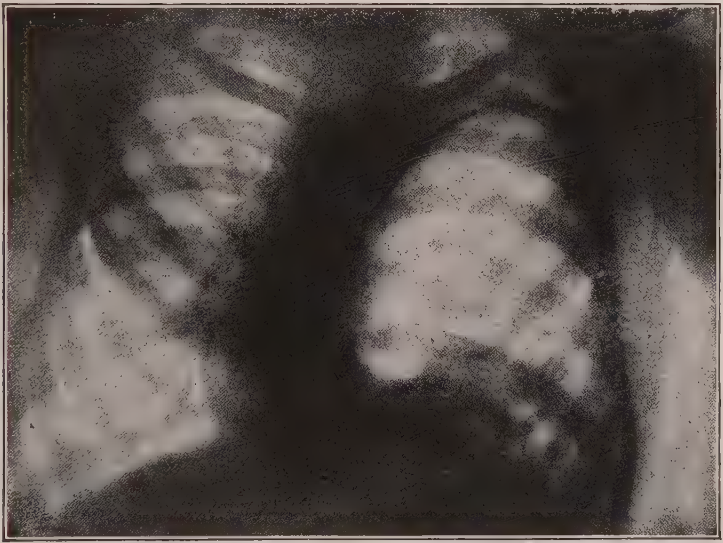

FIG. 189.—Diaphragmatic Hernia. The outline of the displaced diaphragm visible below the left clavicle. Heart displaced to right of sternum. (From v. Ziemssen's Atlas.)

(*a*) The examination of the sputa and of the compressed lung may yield evidence regarding tuberculosis. On the sound side the compensatory hypertrophy covers up foci of dulness or râles so that it is difficult to make out much.

(*b*) Encapsulated pneumothorax gives us practically all the signs of a phthisical cavity, from which it is distinguished by the fact that with a cavity the nutrition of the patient is almost always much worse.

Encapsulated pneumothorax needs no treatment. Hence the importance of distinguishing it from the non-encapsulated form of the disease, in which treatment is essential.

IV. Pleurisy

Clinically, we deal with three types:

(*a*) Dry or plastic pleurisy.
(*b*) Pleuritic effusion, sero-fibrinous or purulent.
(*c*) Pleural thickening (chronic pleurisy).

1. *Dry or Plastic Pleurisy*

Doubtless most cases run their course without being recognized. The frequency with which pleuritic adhesions are found post-mortem would seem to indicate this.

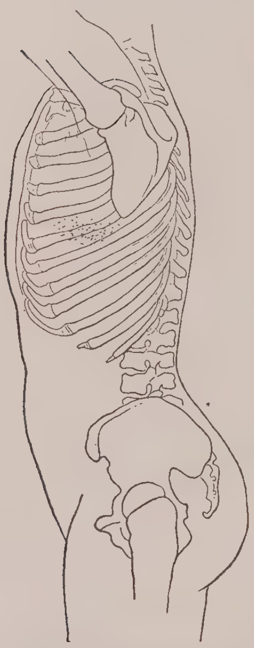

Fig. 190.—Showing the Point at which Pleural Friction is most Often Heard.

It is usually the characteristic stitch in the side which suggests physical examination. The pain and the physical signs resulting from the fibrinous exudation are usually situated at the bottom of the axilla where the diaphragmatic and costal layers of the pleura are in close apposition. Doubtless the pleuritic inflammation is not by any means limited to this spot, but it is here that the two layers of the pleura make the largest excursion while in apposition with each other. In the vast majority of cases, then, the physical signs are situated at the spot indicated in Fig. 190.

Occasionally pleuritic friction is to be heard in the precordial region, and after the absorption of a pleuritic effusion evidences of fibrinous exudation in the upper parts of the chest are sometimes demonstrable. Most rarely of all, evidence of plastic pleurisy may be found at the apex of the lung in connection with early phthisis. In diaphragmatic pleurisy, *i.e.*, when the fibrinous exudation is especially marked upon the diaphragmatic pleura, friction sounds may be heard over the region of the attachment of the diaphragm in front and behind as well as in the axillæ. Hiccup often occurs and gives exquisite pain.

Our diagnosis is based upon a single physical sign, *pleuritic friction*. The nature of this sound has already been described (see above, p. 166), and I will here only recapitulate what was there said. During the first few deep breaths one hears, while listening over the painful area, a grating or rubbing sound usually somewhat jerky and interrupted, most marked at the latter part of inspiration, but often audible throughout the whole respiratory act. After a few breaths it often disappears, but will usually reappear if the patient lies for a short time upon the affected side, and then sits up and breathes deeply. In marked cases the rubbing of the inflamed pleural surfaces may be felt as well as heard, and it is not very rare for the patient to be able to feel and hear it himself. Pleuritic friction may be present and loud without giving rise to any pain. On the other hand, the pain may be intense, and yet the friction-rub barely audible. When heard at the summit of the chest, as in cases of incipient phthisis, pleural friction produces only a faint grazing sound, much more delicate and elusive than the sounds produced at the base of the chest.

Occasionally the distinctive rubbing or grating sounds are more or less commingled with or replaced by crackling sounds indistinguishable from the drier varieties of râles. It is now, I think, generally believed that such sounds may originate in the pleura as well as within the lung. The greatest care should be taken to prevent any shifting or slipping of the stethoscope upon the surface of the chest, as by such means sounds exactly like those of pleural friction may be transmitted to the ear. In case of doubt one should always wet the skin and the stethoscope so that the latter cannot slip.

Muscle sounds are sometimes taken for pleural friction, but they are bilateral, usually low-pitched, sound less superficial than pleural friction, and are not increased by pressure. When listening for friction at the base of the left axilla, I have once or twice been puzzled by some low-pitched rumbling sounds occurring at the end of inspiration, and due (as afterward appeared) to gas in the stomach which shifted its position with each descent of the diaphragm.

The transmitted shoulder-joint crepitus, audible in the back when the patient's arms are crossed in front (F. T. Lord), has been described on p. 151.

In children, friction sounds and pleuritic pain are much less common than in adults, and the signs first recognizable are those of effusion. In adults the presence of a very thick layer of fat may make it difficult or impossible to feel or hear pleural friction.

The breath sounds over the affected area are usually absent or greatly diminished, owing to the restraint in the respiratory movements due to pain. Not infrequently pleuritic friction may be heard altogether below the level of the lung.

2. *Pleuritic Effusion*

Many cases are latent, and the patients consult the physician on account of slight cough, weakness, or gastric trouble, so that the effusion is first discovered in the course of routine physical examination. Since it is usually the results of percussion which first put us on the right track, I shall take up first.

(*a*) *Percussion*

1. *A small effusion* first shows as an area of dulness

(*a*) Just below the angle of the scapula.

(*b*) In the left axilla between the fifth and the eighth rib.

(*c*) Obliterating Traube's semilunar area of tympany; or

(*d*) In the right front near the angle made by the cardiac and hepatic lines of dulness (see Fig. 191).

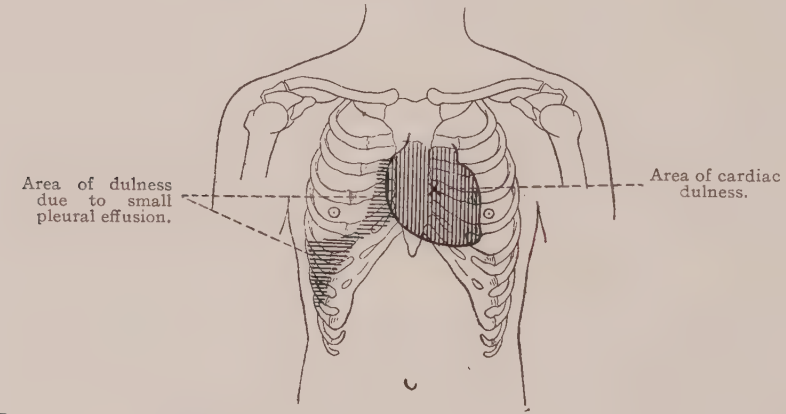

Fig. 191.—Small Pleural Effusion Accumulating (in part) near the Right Border of the Heart.

In the routine percussion of the chest, therefore, one should never leave out these areas. A small effusion is most easily detected in children or in adults with thin chest walls, provided our percussion is not too heavy. An effusion amounting to a pint should always be recognizable, and smaller amounts have frequently been diagnosed and proved by puncture.

The amount of a pleuritic effusion is roughly proportional to the area of dulness on percussion, but not accurately. It is very common to find on puncture an amount of fluid much greater than could have been suspected from the percussion outlines; on the other hand the dulness may be extensive and intense on account of great inflammatory thickening of the costal pleura, by the accumulation of layer after layer of fibrinous exudate and its organization into fibrous plates, while very little fluid remains within.

The amount of dulness depends also upon the thickness and elasticity of the chest wall and the degree of collapse of the lung within.

2. *Large Effusions.*—When the amount of fluid is large, the dulness may extend throughout the whole of one side of the chest with the exception of a small area above the clavicle or over the primary bronchus in front. This area gives a high-pitched *tympanitic* note, provided the bronchi remain open, as they almost always do. This tympany is high-pitched and sometimes astonishingly clear. I recently saw a case in which the note above the clavicle was almost indistinguishable, with the eyes shut, from that obtained in the epigastrium. Occasionally "cracked-pot" resonance may be obtained in the tympanitic area.

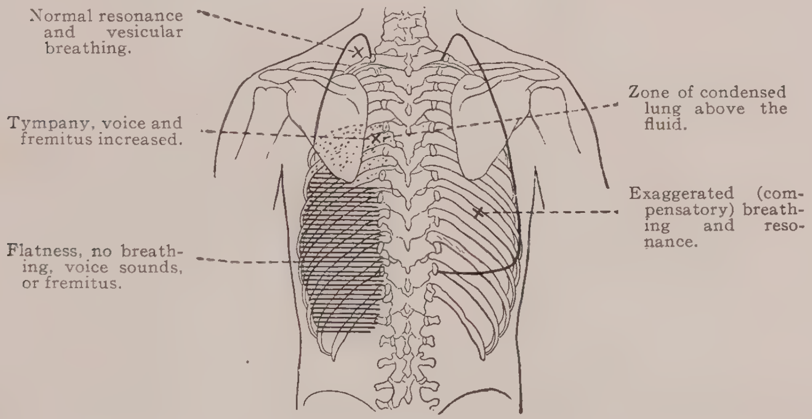
Normal resonance and vesicular breathing.
Tympany, voice and fremitus increased.
Flatness, no breathing, voice sounds, or fremitus.
Zone of condensed lung above the fluid.
Exaggerated (compensatory) breathing and resonance.

FIG. 192.—Diagram to Illustrate Physical Signs in Moderate-sized Effusion in the Left Pleura.

The pitch changes if the patient opens and closes his mouth while we percuss ("William's tracheal tone").

The dulness over the lower portions of a large effusion is usually *very* marked, and the percussing finger feels a greatly increased resistance to its blows when compared with the elastic rebound of the sound side.

3. *Moderate Effusions.*—Three zones of resonance can often be mapped out in the back: at the base dulness or flatness, above that a zone of mingled dulness and tympany, and at the top normal resonance. The lowest zone corresponds to the fluid, the middle zone to

the condensed lung immediately above it, and the top zone to the relatively unaffected part of the lung (see Fig. 192). Not infrequently there is no middle zone but simply dulness below and resonance above, as is usually the case in the axilla and front.

The position of the effusion depends only in part upon the influence of gravity, and is greatly influenced by capillarity and the degree of retraction of the lungs. Consequently the surface of the fluid is hardly ever horizontal. With the patient in an upright position it usually reaches a higher level in the axilla than in the back (see Fig. 193). Near the spine and near the sternum (in right-sided effusions) the line corresponding to the level of the fluid may rise sharply.

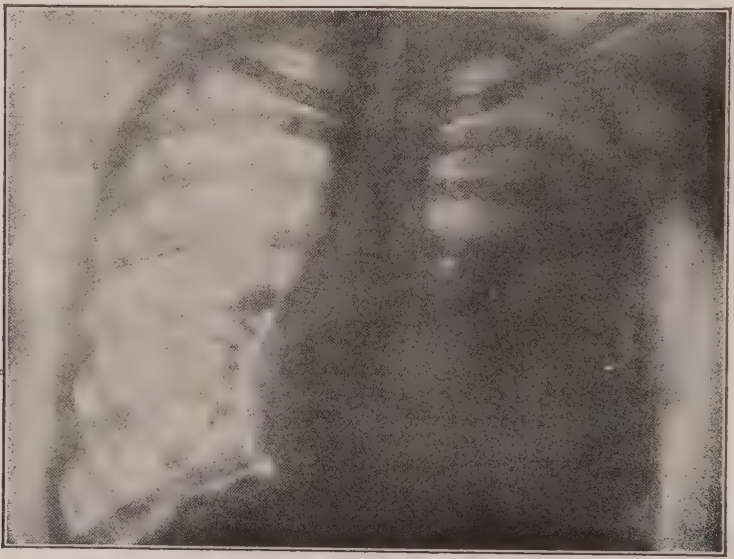

FIG. 193.—Left Pleural Effusion. Heart displaced to right of sternum. Note that the surface of the fluid slopes outward and upward from the median line. (From v. Ziemssen's Atlas.)

There is rarely any information of value to be obtained by trying to elicit a change in the percussion note with change in the patient's position. First, because in the normal chest there is *some* change in the percussion note if we try it first with the patient upright and then bending forward. Next, because even in known pleural effusion it has been found that with change in the position of the patient the level of the fluid sometimes changes very slowly and irregularly, and some times does not change at all. If, for purposes of thorough examination, we raise to a sitting posture a patient who has been for some days or weeks in bed, we should never begin the examination at once, since it may take some minutes for the lungs and the fluid to accommodate themselves to the new position. It is well also to get the patient to cough and to take a number of full breaths before the examination is begun.

At the base of the sound lung behind the sound side a triangular patch of dulness can be percussed out in most cases of pleural effusion. (Grocco's (Koranyi's) Sign—The Paravertebral Triangle.) The voice sound and breath sounds are diminished over this triangular area.

I have not found this sign of much value in diagnosis. One can rarely percuss out the triangular dull area with any confidence unless one previously knows that there is an effusion on the other side.

When the fluid is absorbed or removed by tapping, one would expect an immediate return of the percussion resonance. But in fact the resonance returns very slowly and is wholly unreliable as a test of the amount of absorption which has occurred. Thickened pleura and atelectatic lung may abolish resonance long after the fluid is all gone. We depend here far more upon the evidence obtained by auscultation and palpation and on the general condition of the patient.

To determine the returning elasticity of the lung and the degree of movability of its lower border, percussion is very useful during the stage of absorption. After percussing out the lower border of pulmonary resonance in the back, the patient is directed to take a long breath and hold it. If the lung expands, the area of percussion resonance will increase downward.

Percussion aids us in determining whether neighboring organs are displaced by the pressure of the accumulated fluid. The liver is often pushed down, the spleen *very* rarely. *Dislocation of the heart* is one of the most important of all the signs of pleural effusion, and is often the crucial point in differential diagnosis. It is a very striking and at first surprising fact that a left-sided effusion displaces the heart far more than a right-sided effusion of the same size. Small or moderate right-sided effusions often do not displace the heart at all.

With left-sided effusions, unless very small, we find the area of cardiac dulness shifted toward the right and often projecting beyond the right edge of the sternum (see Fig. 193). (Inspection and palpation often give us even more valuable information on this point. See below, p. 337.) We must be careful to distinguish such an area of dulness at the right sternal margin from that which may be produced in right-sided effusions by the fluid itself (see above).

As mentioned above, a right pleural effusion may very early show itself as an area of dulness along the right sternal margin. Light percussion will usually demonstrate that this dulness is continuous with a narrow strip of flatness at the base of the axilla (ninth and tenth ribs). Such an effusion is late in creeping up the axilla. It appears first and disappars first along the right margin of the sternum.

On the sound side the percussion resonance is often increased, owing to compensatory hypertrophy of the sound lung; the diaphragm is pushed down and the borders of the heart or of the liver may be encroached upon. When the hyperresonance of the sound side is present, it should warn us to percuss lightly over the effusion, else we may bring out the resonance of the distended lung.

Summary of Percussion Signs.—(1) Flatness corresponding roughly to the position of the fluid.

(2) Tympany above the level of the fluid over the condensed lung.

(3) The level of the fluid is almost never horizontal.

(4) Shifting of the fluid with change of position is rare, slow, and has little or no importance in diagnosis.

Exceptions and Possible Errors.—(*a*) Great muscular pain and spasm may produce an area of dulness which simulates that of pleural effusions, especially as the auscultatory signs may be equally misleading. A hypodermic of morphine will dispel the dulness along with the pain if it is due to muscular cramp.

(*b*) Abdominal tumors, ascites, enlarged liver or spleen pushing up the diaphragm into the chest may produce all the signs of pleural effusion. Study of the belly and *x*-ray evidence on the position of the diaphragm should set us right.

(*c*) If the lung on the affected side fails to retract (owing to emphysema or adhesions to the chest wall), the area of dulness and its intensity will be much diminished.

(*d*) It must be remembered that dulness in Traube's space is seldom of significance since it may be due to solidification of the lung, to *situs inversus*, to tumors, or to overfilling of the stomach and intestine with food, as well as to pleural effusion; also that the size of the tympanitic space varies greatly in health.

(*e*) Rarely, percussion may be tympanitic over an effusion at the left base owing to distention of the stomach or colon.

(*f*) The diagnosis between fluid and thickened pleura will be considered later.

(*b*) *Auscultation*

The auscultatory phenomena vary greatly in different cases, and in the same case at different times, because the essential conditions are subject to similar variations. *Whatever sounds are produced in the lungs or in the bronchi may be heard over the fluid unless interfered with by inflammatory thickening of the costal pleura. Fluid transmits sounds well, but there may be no breath sounds produced* and hence none audible over the fluid. Or tubular sounds only may be produced because only the bronchi remain open, the rest of the lung being collapsed.

Or again, if râles or friction sounds are produced in the lung, they, too, may be transmitted to the fluid and may (alas!) deter the timid "observer" from tapping.

In about two-thirds of all large effusions *no* breathing at all is audible over the area of flatness on percussion. In the remaining third, and especially in children, tubular breathing, sometimes feeble, sometimes very intense, is to be heard.

In moderate effusions there are often three zones in the back. At the bottom we hear nothing, in the middle zone distant bronchial or broncho-vesicular breathing, while at the summit of the chest the breathing is normal.

The *voice sounds* correspond. When breath sounds are absent, the voice sounds are likewise absent. When the breathing is tubular, the voice, and especially the whisper, is also tubular and intensified. That is, *whenever the bronchi are open, the lung retracted, and the chest walls thin, the breathing, voice, and whisper will correspond to the tracheal sounds.* Since children have especially thin chest walls, these bronchial sounds are especially frequent and intense in children.

Near the angle of the scapula and in a corresponding position in front, the sound of the spoken voice may have a peculiar high-pitched, nasal twang, to which the term egophony is applied. This sign has no importance in diagnosis, since it is not constant, and not peculiar to fluid accumulations.

Râles are rarely produced in the retracted lung, and so are rarely to be heard over the fluid.

All these sounds may be diminished or abolished if the costal pleura is greatly thickened.

The influence of cough upon the lung, and so upon the sounds produced in it and transmitted through the fluid, may be very great and very puzzling. Râles may appear or disappear, breathing change in quality or intensity. Hence in the differential diagnosis of difficult cases the patient should always be made to cough and then breathe deeply before the examination is completed.

In very large effusions, when only the primary bronchi are open, there may be signs like those of pulmonary cavity at the site of the bronchi in front or behind (amphoric breathing, large metallic râles, etc.). Over the sound lung the breathing is exaggerated and extends unusually far down in the back and axilla, owing to hypertrophy of the lung.

The heart sounds may be absent at the apex owing to dislocation of the heart. In left-sided effusions, the apex sounds are often loudest near the ensiform cartilage or beyond the right margin of the sternum. Right-sided effusions have much less effect upon the heart, but occasionally we find the heart sounds loudest at the left of the nipple or in the axilla.

Since most cases of pleural effusion are due to tuberculosis, we should never omit to search for evidences of this disease at the apex of the lung on the *sound* side, since experience has shown that phthisis is more apt to begin here than on the side of the effusion.

Summary of Auscultatory Signs

(1) In most cases, voice and breath sounds are absent or very feeble over the area occupied by the fluid.

(2) In a minority of the cases, the breathing and voice sounds may be tubular and intensified, especially in children.

(3) Over the condensed lung at the summit of the chest, the breathing is bronchial or broncho-vesicular, according to the degree of condensation. If the amount of fluid is small, the layer of condensed lung occupies the middle zone of the chest and the breathing is normal at the top of the chest.

(4) Râles and friction sounds are rarely heard over fluid.

(5) On the sound side the breathing is exaggerated.

(6) The heart sounds may be absent at the apex and present in the left axilla or to the right of the sternum owing to dislocation of the heart.

(*c*) *Inspection and Palpation*

The most important information given us by inspection and palpation relates to the displacement of various organs by the pressure of the accumulated fluid. In left-sided pleuritic effusions the heart is usually displaced considerably toward the right, even when the level of the fluid reaches no higher than the sixth rib in the nipple line. The impulse is then to be seen and felt to the right of the sternum, somewhere between the third and the seventh rib, when a large amount of fluid is present. With smaller effusions one may find the apex beat lifting the sternum or close to its left border. The position of the heart may be confirmed by percussion.

The spleen is scarcely ever displaced.

Right-sided effusions are far less likely to displace the heart, and it is only when a large amount of fluid is present that the apex of the heart is pushed outward beyond the nipple. Moderate right-sided effusions often produce no dislocation of the heart whatever. The liver may be considerably pushed down by a right-sided pleuritic effusion, and its edge may be palpable several inches below the costal margin. Its upper margin cannot be determined by percussion, as it merges into the flatness produced by the fluid accumulation above it.

Tactile fremitus is almost invariably absent or greatly diminished over the areas corresponding to the fluid; just above the level of the fluid it is often increased. Spasm of the intercostal muscles near the fluid can often be felt and may account for part of the percussion dulness.

Occasionally a slight fulness of the affected side may be recognized by inspection, and the interspaces may be less readily visible than upon the sound side. Bulging of the interspaces I have never observed. When the accumulation of fluid is large the respiratory movements upon the affected side are somewhat diminished,[1] the shoulder is raised, and the spine curved toward the affected side. The diaphragm is depressed, and Litten's sign therefore absent.

There are no reliable physical signs for distinguishing purulent from serous effusions. The whispered voice may be transmitted through either pus or serum. But we know that in children two-thirds of all effusions are purulent (pneumococcus or streptococcus infection), while in adults three-fourths of them are serous (tuberculosis of the pleura).

Physical Signs During Absorption of Pleural Effusions

When the fluid begins to disappear, either spontaneously or as a result of treatment, the dulness very gradually disappears and the breath sounds, voice sounds, and fremitus reappear. In case the heart has been dislocated, its return to its normal position is often much slower than one would anticipate, and indeed all the physical signs are disappointingly slow to clear up even after tapping. Pleural friction appears when the roughened pleural surfaces, which have been held apart by the fluid, are allowed by the disappearance of the latter to come into apposition again. Owing to pulmonary atelectasis and permanent thickening of the pleura, considerable dulness often remains for weeks after the fluid has been absorbed. One need not be disappointed or believe that the fluid has again accumulated if one finds but little change in the physical signs during the first week after tapping. A second puncture rarely shows fluid.

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[1] I have purposely made but little of the changes in the shape of the chest produced by pleuritic effusions, as it has seemed to me that by far too much stress has usually been laid upon such signs.

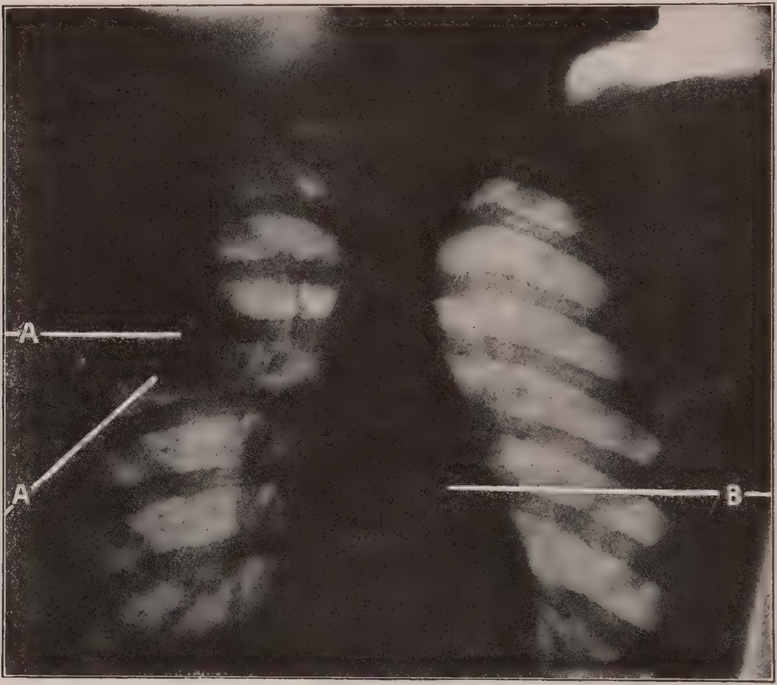

FIG. 194.—Interlobar Empyema. A, A = Exudate. B = Heart. (Englebach and Carman.)

(d) *Interlobar Empyema*

In recent years the frequency and importance of empyema limited to an interlobar fissure has become impressed upon many clinicians.

One sees both the post-pneumonic and the tuberculous types, but the former is vastly the commoner. In most of the cases so far reported the pus has been demonstrated in the fissure which runs along the vertebral border of the scapula when that bone is pulled as far forward as possible by crossing the arms in front (see Fig. 195).

This is a region seldom carefully examined.

In the strip here indicated one finds flatness on percussion with (usually) diminished fremitus, and feeble or absent breath-sounds.

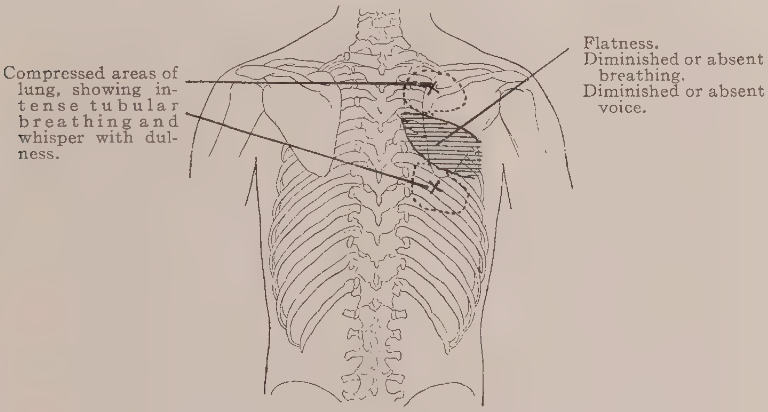
Compressed areas of lung, showing intense tubular breathing and whisper with dulness.
Flatness.
Diminished or absent breathing.
Diminished or absent voice.

FIG. 195.—Signs in Interlobar Empyema.

X-ray examination may bring out in sharp relief a shadow corresponding to this area and sharply contrasted with the relatively normal lung above and below it (see Fig. 194).

The exploring needle often fails to find the pus, but the *search should not be given up* (*if the physical signs are clear*) *until a rib has been excised and the region thoroughly explored under complete anæsthesia.* Empyema encysted between the diaphragm and lung, between the lung and chest wall, and along the right or left cardiac borders are not uncommon but can rarely be diagnosed.

3. *Pleural Thickening*

In persons who have previously suffered from pleurisy with effusion, and in many who have never to their knowledge had any such trouble, a considerable thickening of the pleural membrane with adhesion of the costal and visceral layers may be manifested by the following signs:

(1) Dulness on percussion, sometimes slight, sometimes marked.

(2) Diminished vesicular respiration.

(3) Voice sounds and tactile fremitus diminished or increased.

(4) Absence of Litten's phenomenon and diminution in the normal respiratory excursion of the chest.

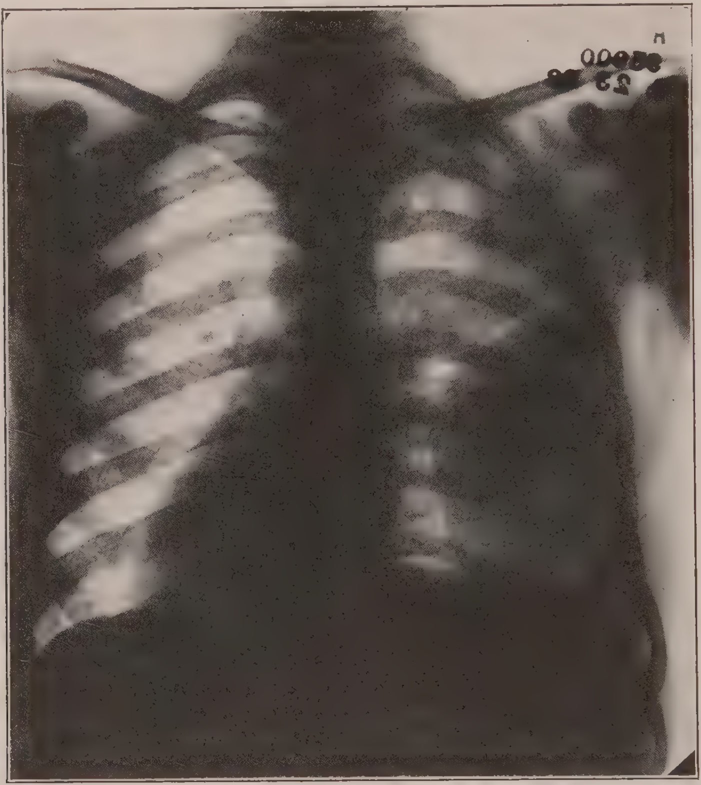

FIG. 196.—Encapsulated empyema at right base.

These signs are most apt to be found at the base of the lung behind and in the axilla. Occasionally a similar thickening may be demonstrated throughout the whole extent of the pleura, and the lung failing to expand, the chest may fall in as a result of atmospheric pressure.

The ribs approximate and may overlap, the spine becomes curved, the shoulder lowered, the scapula prominent, and the whole side shrunken. The heart may be drawn toward the affected side.

In the diagnosis of pleural thickening, Rosenbach's "palpatory puncture" is sometimes our only resource. Under antiseptic precautions a hollow needle is pushed between the ribs and into the pleural cavity. As the needle forces its way through the tough fibrous, or perhaps calcified, pleura, the degree and kind of resistance are very enlightening. Again, the amount of mobility of the point after the chest wall has been pierced tells us whether the needle is free in a cavity, entangled in a nest of adhesions, or fixed in a solid "carnified" lung. There is no danger if the needle is sterile.

Diffuse neoplasm spreading over the whole pleura may give all the signs of thickened pleura, though usually with rapidly recurring effusion. This disease may be mistaken for chronic pleurisy even *post mortem* unless histological examination is expert.

Hæmorrhagic effusions, pain after tapping, and progressive cachexia may put us on the track of the diagnosis. Occasionally metastatic glands are found in the brain, neck or axillæ. This disease must always be remembered in the differential diagnosis of thickened pleura.

4. *Encapsulated Pleural Effusion*

Small accumulations of serum or pus may be walled off by adhesions so that the fluid does not gravitate to the lowest part of the pleural cavity or spread itself laterally as it would if free. Such localized effusions are not uncommon. Thirty-one per cent of thirty-eight empyemata autopsied at the Massachusetts General Hospital were encapsulated. They are most apt to be found in the lower axillary regions or behind—sometimes between the base of the lung and the diaphragm, and more often between the lobes of one of the lungs or higher up. The position of the fluid may be almost vertical, lying in a shallow pool along the axillary ribs or near the spinal column. I have twice seen an encapsulated purulent effusion so close to the left margin of the heart that the diagnosis of pericardial effusion was made.

The diagnosis of encapsulated pleural effusion is a difficult one and often times cannot be made except by puncture. The signs are those of fluid in the pleura, but anomalously placed. Even puncture may fail to clear the difficulty, since the needle may pass entirely through the pouch of fluid and into some structure behind so that no fluid is obtained.

5. *Pulsating Pleurisy*

Under conditions not altogether understood, the movements transmitted by the heart to pleural effusion (usually purulent) may be visible externally as a circumscribed pulsating swelling near the precordial region, or as a diffuse undulation of a considerable portion of the chest wall. Sometimes this pulsation is visible because the fluid has worked its way out through the thoracic wall and is covered only by the skin and subcutaneous tissues, but occasionally pulsation in a pleural effusion becomes visible, although no such perforation of the chest wall has occurred.

The condition is a rare one (only 12 cases are on record), and is of importance only because it may be mistaken for an aneurism, from which, however, it should be readily distinguished by the history, the absence of a palpable thrill or diastolic shock and by the evidence of fluid in the pleura.

6. *Differential Diagnosis of Pleuritic Effusion*

The following conditions are not infrequently mistaken for pleuritic effusion:

(1) Croupous pneumonia with occlusion of the bronchi.
(2) Pleural thickening with pulmonary atelectasis.
(3) Subdiaphragmatic abscess of the liver.

In croupous pneumonia with plugging of the bronchi one may have present all the physical signs of pleuritic effusion *except displacement of the neighboring organs*. The presence or absence of such displacement, together with the history, symptoms, and course of the case, is therefore our mainstay in distinguishing the two diseases.

From *ordinary* croupous pneumonia (without occlusion of the bronchi) pleuritic effusion differs in that it produces a greater degree of dulness and a diminution of the spoken voice sounds and tactile fremitus. Bronchial breathing and bronchial whisper may be heard either over solid lung or over fluid accumulation, although the bronchial sounds are usually feeble and distant in the latter condition. The displacement of the neighboring organs is of importance here as in all diagnoses in which pleuritic effusion is a possibility. A few hard coughs may open up an occluded bronchus and so clear up the diagnosis at once. In doubtful cases the patient should always be made to cough and breathe deeply before the examination is finished.

It should always be remembered that one may have both pneumonia and pleuritic effusion at the same time, and that pneumonia is often accompanied by a serous, or followed by a purulent, effusion. In children the bronchi are especially prone to become occluded even as a result of a simple bronchitis, and we must then differentiate between atelectasis and effusion—in the main by puncture and by the use of the criteria just described.

(2) It is sometimes almost impossible to distinguish small fluid accumulations in the pleural cavity from pleural thickening with pulmonary atelectasis. In both conditions one finds dulness, diminution of the voice sounds, respiration, and tactile fremitus, and absence of Litten's phenomenon, but the tactile fremitus is usually more diminished when fluid is present than in simple pleural thickening and atelectasis. The presence of friction sounds over the suspected area speaks strongly in favor of pleural thickening, but it is possible to hear friction sounds over fluid, probably because they are conducted from a point higher up in the chest at which no fluid is present. In doubtful cases the diagnosis can and should be cleared up by *puncture*.

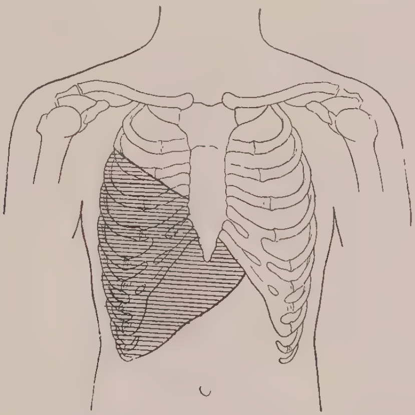

Fig. 197.—Area of Dulness in Solitary (tropical) Abscess of the Liver.

(3) In several cases I have known subdiaphragmatic abscess or an enlargement of the liver due to multiple abscesses to be mistaken for empyema. In both conditions, one finds in the right back, dulness on percussion as high as mid-scapula, with absence of voice sounds, breath sounds, and fremitus. These conditions are due in one case to the presence of fluid between the lung and the chest wall, and in the other case to the liver or abscess fluid which pushes up the lung together with the diaphragm. Without the fluoroscope or a good radiograph this diagnosis may be impossible. With the fluoroscope

it should be possible to see that the dome of the diaphragm caps the shadow and moves but slightly during inspiration. Some of the symptoms, such as chills, sweating, and irregular fever, are common to both conditions. A careful consideration of the history and the associated signs and symptoms may help us to decide.

Large solitary abscess of the liver, occurring as it almost invariably does in the posterior portions of the right lobe, produces an area of flatness or percussion, which rises to a much higher level in the axilla and back than in front or near the sternum (see Fig. 198), and may be in this way distinguished from empyema; but when the liver contains many small abscesses, as in suppurative cholangitis, this peculiar line of dulness is not present.

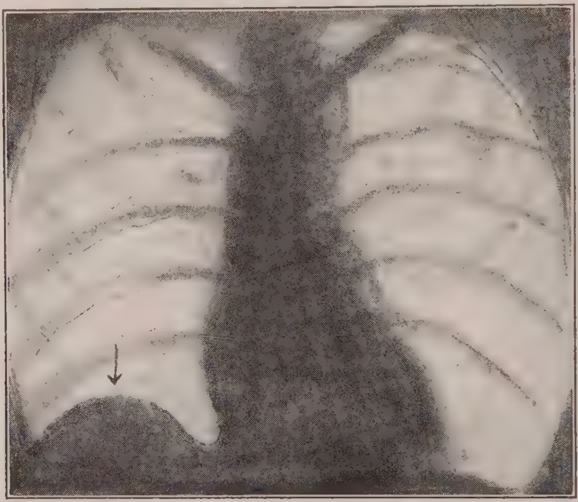

FIG. 198.—Pencil sketch of an x-ray plate showing (see the arrow) a diaphragm domed over a subdiaphragmatic abscess.

(4) Rare diseases, such as cancer or hydatid of the lung, may be mistaken for pleuritic effusion. The history of the case and the results of exploratory puncture usually clear up the difficulty.

7. *Carcinoma of the Pleura* (*Endothelioma*)

About fifty cases are on record. Probably many are falsely diagnosed as a chronic rapidly refilling pleural effusion. The fluid obtained by tapping is usually bloody either from the first or later, and contains a larger proportion of endothelial plaques and a smaller proportion of lymphocytes than is usual in chronic pleurisy. The cells themselves, however, are not characteristic of a neoplasm.

The physical signs in the chest do not differ from those of any ordinary pleural effusion. The points of differential diagnostic value are:

1. The presence of bloody fluid with an endothelial sediment.
2. Its rapid and repeated reaccumulation.
3. Metastases (cerebral, supraclavicular, axillary, pectoral and along the needle-track).
4. Pain after tapping.

Similar signs are obtained in the rare sarcoma of the pleura (fourteen cases on record).

8. *Cancer of the Bronchi and Lung*

The condition is almost impossible to diagnose. But 10 of the 90 cases collected by Weller (Arch. of Int. Med., 1913, XI, 314) were recognized in life; 35 of 42 cases in this group had hæmoptysis (sputa *not* characteristic). Thirteen per cent. had brain metastasis. The other signs are pain, cough dyspnœa and other evidence of bronchial obstruction, pressure symptoms due to regional metastases and the absence of fever.

9. *Echinoccocus of the Pleura*

This disease is almost unknown in North America. Forty-three foreign cases are on record. The signs are those of encysted pleural fluid containing eosinophiles and hooklets, remarkable in that it *does not contain albumen* in any considerable quantity. Urticaria often follows puncture.

10. *Actinomycosis of the Pleura*

There are no characteristic physical signs. These diseases may be suspected if an empyema perforates the chest or is associated with chronic pulmonary suppurating. Diagnosis depends on the microscopic examination of the fluid.

V. Cyto-Diagnosis of Pleural and Other Fluids

Only such methods as can be carried out without a thermostat will be here described. Hence the examination of diphtheria swabs, blood cultures, and pus are excluded. We have left the fluids obtained by tapping the pleura, the peritoneum, and the spinal cord. The first is the most important.

Pleural Fluids.—A fluid withdrawn from the pleura by puncture may be a mechanical transudate (hydrothorax), may be evidence of *tuberculous pleurisy* (primary or associated with phthisis), or, rarely, an exudate of septic or cancerous origin.

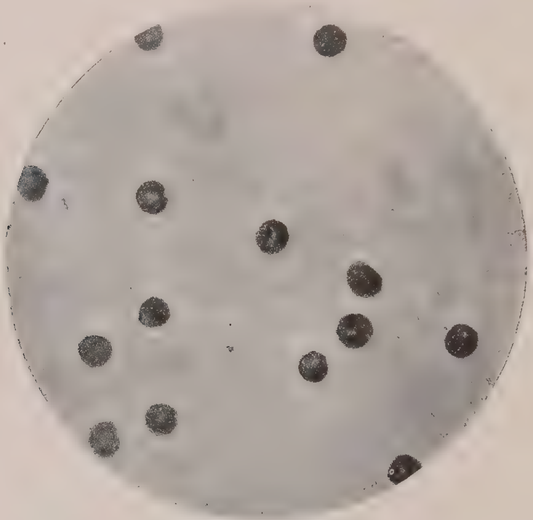

Fig. 199.—Lymphocytosis in Pleural Fluid. Primary tuberculous pleurisy. (×750 diameters.) (Musgrave.)

To investigate these fluids we note:

1. *Color.* Bloody fluids suggest cancer, but occasionally occur in pneumonia and tuberculosis.

2. *Weight.*[1] Dropsical fluid is generally *below* 1.015 in specific gravity. Exudates are usually in the vicinity of 1.020. An ordinary specific-gravity bulb is used.

3. *The cells of the sediment* (cytodiagnosis).

1. *Technique of Cytodiagnosis*

Pour fluid into tubes of a centrifuge and centrifugalize five minutes.

2. Pour off the supernatant fluid and stir up the sediment with a platinum loop, so as to suspend the sediment in the few remaining drops.

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[1] The amount of albumin usually runs parallel with the weight of the fluid.

3. Spread a drop of the mixture on a *clean* cover glass with the platinum loop and let the smear dry without heating it.

4. Stain like a blood film (see below, page 466) with the following mixture:[1] Wright's modification of Leishman's stain, 3 parts; *pure* methyl alcohol, 1 part.

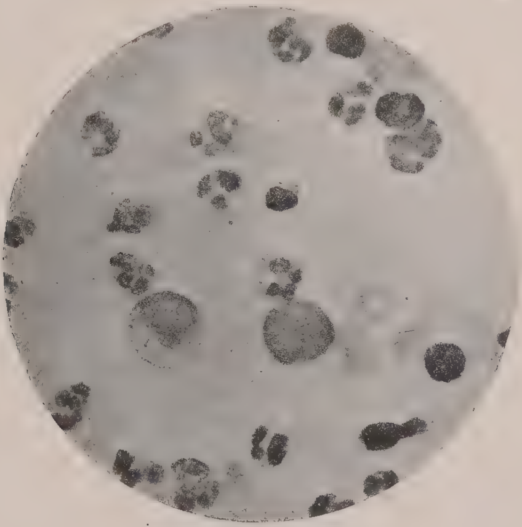

FIG. 200.—Polynuclears and Large Lymphocytes in Pleural Fluid from a Case of Traumatic Acute Infectious Pleurisy. (×750 diameters.) (Musgrave.)

5. After staining, wash *very* gently, using a dropper (else the whole film may be pushed off), and dry *in the fingers* over a Bunsen or alcoholic flame. Do not blot the preparation.

6. Mount in Canada balsam and examine with an oil-immersion lens.

2. *Interpretation of Results*

(*a*) In *tuberculous pleurisy*, *lymphocytes* make up from seventy to ninety-nine per cent—usually over ninety per cent—all of the cells found in the smear[2] (see Fig. 199).

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[1] Suggested by Musgrave: Boston Med. and Surg. Journ., vol. cli., p. 319, 1904.

[2] This rule, however, does not work both ways. Tuberculosis produces lymphocytosis, but so do other chronic irritations. *The lymphocytosis is a mark of chronicity and only suggests tuberculosis*, but there are no other common causes for chronic pleural irritation.

(*b*) In septic cases due to the streptococcus, staphylococcus, or pneumococcus the majority of the cells are *polynuclear* leucocytes (see Fig. 200).

(*c*) In *transudations* (dropsical) the predominating cell is a large mononuclear type, apparently endothelial in origin and other occurring in sheets or "*plaques*" (see Fig. 201).

Exceptions occasionally occur, but in the main these rules are sufficiently exact to be of value in diagnosis when taken in connection with all the facts in the case.

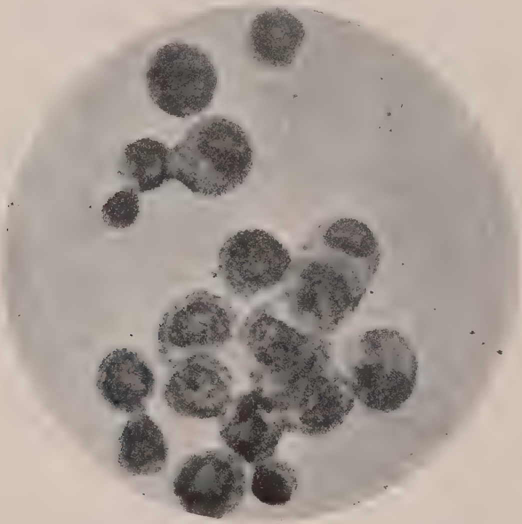

Fig. 201.—Pleural Fluid in Hydrothorax due to Cardiac Disease. Endothelial plaques and cells. (×750 diameters.) (Musgrave.)

In *peritoneal fluid* the use of cytodiagnosis has not as yet furnished information of any considerable diagnositic value.

In cerebrospinal fluid obtained by lumbar puncture the predominance of lymphocytes is not so often associated with tuberculosis as it is in the pleura, but usually means chronic cerebrospinal irritation such as is produced by dementia paralytica and tabes. As excess of polynuclear cells is usually due to acute meningitis—epidemic or sporadic.

CHAPTER XX

ABSCESS, GANGRENE, AND CANCER OF THE LUNG, PULMONARY ATELECTASIS, ŒDEMA, AND HYPOSTATIC CONGESTION

I. Abscess and Gangrene of the Lung

I consider these two affections together, because the physical signs, exclusive of the sputa, do not differ materially in the two affections. In some cases there may be no physical signs at all, and the diagnosis is made from the character of sputa and from a knowledge of the etiology and symptomatology of the case. In 100 cases studied by Lord at the Massachusetts General Hospital, 22 were due to pneumonia. Inhalation of septic material (as during tonsil operations), extension from the pleura, bronchi or subdiaphragmatic region, septic emboli and trauma are the next most frequent causes.

The right lower lobe close beneath the pleura is a favorite site. In 25 of Lord's 38 cases there were multiple abscesses. Acute abscess may be single. Chronic infections are generally multiple. The pleura is firmly adherent in only one-third of the cases. The symptoms are of sepsis with cough and purulent, foul, sometimes bloody sputa, containing elastic tissue. In most cases we find nothing more than a patch of coarse râles or a small area of solidification, over which distant bronchial breathing, with increased voice sound and fremitus, may be appreciated. Usually there is some localized dulness on percussion. One may find the signs of cavity (amphoric breathing, cracked-pot resonance, and gurgling râles), but this is unusual.

Pulmonary abscess is often simulated by the breaking of an empyema into the lung and the emptying of the pus through a bronchus. Large quantities of pus are expectorated in such a condition, and abscess of the lung is suggested, but the other physical signs are those of *elastic fibres* is the crucial point in the diagnosis of intrapulmonary abscess, whether due to the tubercle bacillus or to other organisms. Tuberculous abscess (cavity) is usually near the summit of the lung, and other varieties of abscess are near the base, but often there are no physical signs by which we can distinctly localize the process.

II. Neoplasms of the Lung and Mediastinum

Neoplasms of the lung are usually secondary to tumors of the digestive tract, bones, uterus or breast, and are recognized chiefly by the presence of ill-defined pulmonary symptoms in patients known to have previously suffered from neoplasms elsewhere in the body.

Primary neoplasms, 87 per cent. of which start in the bronchi, may be quite without physical signs. The recognizable cases fall into two groups:

1. The lung type.
2. The mediastinal pressure type.

In the pulmonary type neoplasms reaching near to the pleural surface of the lung may produce a diminution or loss of percussion resonance, breath sound, voice, and fremitus. Diagnosis is usually made by *x*-ray alone. The disease is usually mistaken for lung abscess, bronchiectasis or tuberculosis.

In the mediastinal type the diagnosis of mediastinal tumor is usually made. Metastatic glandular masses produce pressure signs like those of primary mediastinal neoplasms.

1. *Mediastinal Neoplasms*

According to Christian,[1] the mediastinal neoplasms which are neither so rare nor so obscure as to make diagnosis practically impossible are: (1) Sarcoma (including lymphosarcoma, leucæmic growths, and Hodgkins' disease); (2) Teratoma and cyst.

2. *Mediastinal Sarcoma*

Starting in the local lymph nodes, in the thymus or in the connective tissue, occurring at any age and chiefly in males, the growths comprise neighboring structures and thus produce dyspnœa, cough, and pain, sometimes dysphagia and hoarseness.

The physical signs are: (a) prominence or bulging of the regions near the manubrium; (b) distension of the veins of the neck and upper thorax, cyanosis and localized œdema from pressure on the cava or its branches; (c) metastatic tumors in the neck which may push the trachea to one side; (d) percussion dulness on each side of the manubrium with diminished vocal and tactile fremitus. Auscultation rarely yields characteristic results, though there may be noisy strident breath-sound from pressure on a bronchus. (e) Evidence of pleural effusion. (f) *X*-ray shadows of characteristic irregular shape.

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[1] Christian: Osler's Modern Medicine, vol. iii, p. 893.

3. *Differential Diagnosis*

Our chief business is to exclude aneurism. This is usually possible by studying the shape of the *x*-ray shadow, the course and history of the case, the Wassermann reaction, and the pressure symptoms which with tumors are far more apt to include venous distension, œdema, and cyanosis than is the case with aneurism.

4. *Cysts of the Mediastinum*

Christian (*loc. cit.*) has collected sixty-four cases of dermoid cyst or teratoma of the mediastinum. Most cases occur before the thirtieth year. The course is very chronic. The cyst may exist for years without producing any symptoms and then be accidentally discovered in the course of an *x*-ray examination undertaken for some other purpose. When it grows large enough to produce pressure symptoms it may give rise to dyspnœa, pain, and cough. In eleven of Christian's cases *hair was expectorated* as a result of communication between the cyst and a bronchus. Bulging of the chest wall near the manubrium with dulness on percussion, diminished breathing, and vocal sounds, and an often characteristically spherical ovoid *x*-ray shadow, are the most constant physical signs.

III. ATELECTASIS

(*a*) Areas of atelectasis or collapse of pulmonary tissue are often present in connection with various pathological processes in the lung (such as tuberculosis or lobular pneumonia), but are usually too small to give rise to any characteristic physical signs; nevertheless

(*b*) In most normal individuals a certain degree of atelectasis of the margins of the lungs may be demonstrated in the following way: The position of the margins of the lungs in the axillæ, in the back, or in the precordial region are marked out by percussion at the end of expiration. The patient is then directed to take ten full breaths, and the pulmonary outlines at the end of expiration are then percussed out a second time. The pulmonary resonance will now be found to extend nearly an inch beyond its former limits, owing to the distention of previously collapsed air vesicles.

If one auscults the suspected areas during the deep breaths which are used to dispel the atelectasis, very fine râles are often to be heard at the end of expiration, disappearing after a few breaths in most cases, but sometimes audible as long as we choose to listen to them. These sounds, to which there is given the name of "atelectatic crepitation," are, in my experience, especially frequent at the base of either axilla. The same writer has noticed an opacity to the *x*-rays over such atelectatic areas.

Forcible percussion may be sufficient to distend small areas of collapsed lung, or at any rate to dispel such dulness as has been previously present.

(*c*) When one of the large bronchi is compressed, as by an aneurism, or occluded by a foreign body, collapse of the corresponding area of lung may be shown by diminished motion of the affected side, dulness on percussion, and absence of breathing, voice sounds, and tactile fremitus.

In new-born babies whose lungs do not fully expand at the time of birth, similar physical signs are present over the non-expanded lobes. The right lung is especially apt to be affected.

In the differential diagnosis of extensive pulmonary collapse, the etiology, the suddenness of the onset, the absence of fever and of displacement of neighboring organs enable us to exclude pneumonia and pleuritic effusion. In distinguishing small areas of solidification from similar areas of atelectasis fluoroscopic studies may be of value. Atelectatic areas brighten up with cough and deep breathing. Solidified areas show no change.

IV. Œdema of the Lungs

(*a*) *Chronic Form.*—In cardiac or renal disease one can often demonstrate that the lungs have been invaded by transuded serum as a part of the general dropsy. More rarely, pulmonary œdema exists without much evidence of œdema in other organs or tissues.

The only physical sign characteristic of this condition is the presence of numerous râles in the dependent portions of the lungs; that is, throughout their posterior surfaces when the patient has been for some time in a recumbent position; or over the lower portions of the axillæ and the back if the patient has not taken to his bed.

The râles are always bilateral (unless the patient has been lying for a long time on one side), and the individual bubbles appear to be all of the same size, or nearly so, differing in this respect from those to be heard in bronchitis. Squeaking or groaning sounds are less often heard. The respiratory murmur is usually somewhat diminished in intensity.

Dulness on percussion and modification of voice sounds are not present, unless hydrothorax or hypostatic pneumonia complicate the œdema.

(*b*) *Acute Form.*—In brain lesions, comatose conditions, and hypertensive cardio-vascular disease the lungs may suddenly fill with fluid "out of a clear sky" and with such urgency that pinkish frothy serum pours from the mouth or is rapidly expectorated. The patient may die in the first attack, or often in later attacks. The physical signs are coarse bubbling râles throughout the lungs with feeble heart.

V. HYPOSTATIC PNEUMONIA

In long-standing passive congestion, the alveoli of the dependent portions of the lungs may become so engorged with blood and alveolar cells as to be practically solidified. Under these conditions, examination of the posterior portions of the lungs shows usually:

(*a*) Slight dulness on percussion reaching usually from the base to a point about one-third way up the scapula. At the very base the dulness is less marked and becomes mixed with a shade of tympany.

(*b*) Feeble or absent tactile fremitus.

(*c*) Diminished or suppressed breathing and voice sounds.

The right lung is apt to be more extensively affected than the left.

Occasionally the breathing is tubular and the voice sounds are increased, making the physical signs identical with those of croupous pneumonia, but as a rule the bronchi are as much engorged as the alveoli to which they lead, and hence no breath sounds are produced.

Râles of œdema or of bronchitis may be present in the adjacent parts of the lungs. The fact that the dulness is less marked at the base of the lung than higher up helps to distinguish the condition from hydrothorax.

The diagnosis is usually easy, owing to the presence of the underlying disease. Fever, pain, and cough such as characterize croupous pneumonia are usually absent.

CHAPTER XXI

THE ABDOMEN IN GENERAL, THE BELLY WALLS, PERITONEUM, OMENTUM, AND MESENTERY

I. Examination of the Abdomen in General

Our methods are crude and inexact compared to those applicable to the chest. Auscultation, despite Cannon's brilliant foundation studies,[1] is of practically no use. Inspection is helpful in but few cases. Palpation, our mainstay, is often rendered almost impossible by thickness, muscular spasm, or ticklishness of the abdominal walls. Percussion is of great value in some cases, but yields no useful results in the majority.

1. *Technique*

The knack of abdominal examination, and especially that part of it whereby the skilled diagnostician gets his most valued information, is difficult even to demonstrate and almost impossible to describe. Hence the account of it in this and other books is very brief when compared with the space allotted to the methods of examining the chest.[2]

The table or bed on which the patient lies during most abdominal examinations (excluding gynæcological work) should be at least three feet *high*, narrow, and *firm*. Most beds are too low, too wide, and too soft; but, on the other hand, the patient must not be made uncomfortable by the hardness or coldness of the surface on which he lies. A comfortable pillow should be provided.

(*a*) *Inspection*

We need a tangential light, such as accentuates by shadows every unevenness of the surface. If the patient is examined in the ordinary dorsal decubitus, the light from any single window, except one overhead, is satisfactory. If one inspects the abdomen with the patient upright, he should stand with his side to the light, not facing it. By inspection we seek information on:

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[1] Summarized in his "Mechanical Factors of Digestion:" Longman's, 1911.

[2] I have heard a physician in a leading American city say that when palpation of the spleen in typhoid fever was first introduced, there was but one physician in the city who had the knack, and that his colleagues were very sceptical about the possibility of accomplishing the feat at all. I have seen a similar uncertainty regarding the palpation of the normal but slightly displaced right kidney.

(*a*) The general contour of the abdomen.
(*b*) The surface of the belly walls, especially the skin and the navel.
(*c*) Respiratory movements, their limitation or absence.
(*d*) Peristaltic movements (gastric or intestinal in origin).
(*e*) The presence of local prominence or (rarely) depression.

Inspection of the Belly Wall.—1. The *surface of the belly wall* is often searched most carefully for the *rose spots* of typhoid fever, which are hyperæmic, very slightly elevated spots, about the diameter of a large pin head (2–4 mm.). They disappear on pressure. Pimples are usually larger, better defined at the edges, and more highly colored, contrasting with the very *pale red* of most rose spots. They are by no means confined to the belly and may be found exclusively on the back. Having been at the outset somewhat sceptical of their value in diagnosis, I have become thoroughly convinced by greater experience and more careful examination. Richardson[1] has shown that they often contain bunches of typhoid bacilli. The spots are present in about three-fourths of all cases, and, while they also may occur in any disease when the blood contains bacteria (*e.g.*, sepsis), they are commonest in typhoid.

2. Distended and tortuous veins on the abdomen are seen in diseases obstructing the portal circulation (rarely in cirrhotic liver) or the inferior cava.

3. Striæ, or linear markings on the skin of the abdomen, follow the subsidence of any long-standing trouble that stretches the skin—pregnancy, obesity, tumors, etc. They are red, are angry when first produced, but later turn white (*lineæ albicantes*).

4. Scars of old wounds or operations may be of great diagnostic value in comatose or delirious cases.

5. Projection or levelling of the normal depression at the navel is evidence of distention, usually by fluid, within the belly.

Respiratory movements of the belly walls are limited or cease in painful diseases within the peritoneum (peritonitis, lead colic) or when the diaphragm is pushed up by a large tumor, ascites, or meteorism.

Peristaltic waves creeping along beneath the belly walls are seen with chronic stenosis and obstruction at the pylorus or at some point in the colon and occasionally in *thin* but healthy persons.

Herniæ and local and general prominences will be discussed in connection with abdominal tumors.

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[1] M. W. Richardson: Pennsylvania Medical Journal, March 3, 1900.

(*b*) *Palpation*[1]

With the patient on the back upon a suitable bed or table,[2] the head on a comfortable pillow, and the abdomen exposed, run the palm of the hand (warmer) lightly over the whole surface, to accustom the muscles to its presence. Then try whether better relaxation of the belly walls is obtained when the patient's knees are drawn up. Some patients relax better in this position; others when the legs are extended.

If the muscles of the abdomen remain contracted and stiff even when the patient is comfortable and has become accustomed to the presence of the physician's hand, we may try to induce relaxation:

(*a*) By getting the patient to take a series of deep breaths.

(*b*) By diverting his attention through conversation or otherwise.

If these means fail and it is important that we should thoroughly investigate the abdomen, we have left two further ways of producing relaxation, viz.:

(*c*) By putting the patient into a warm bath.

(*d*) By anæsthesia (ether or chloroform).

The movements of the physician's hand should never be sudden or rough. He should avoid digging into the skin with his nails or pressing strongly on a small spot with the finger-tips. If any spot be suspected to be tender, that should be palpated last, after going over the rest of the abdomen. If it is necessary to make deep pressure at any point, it is best to lay the fingers of the left hand loosely over the spot and then exert pressure upon them with the fingers of the right hand. The passive hand is more sensitive. To reach a deep spot, put the hands in this position over it, ask the patient to take a long breath, and, as the belly falls in expiration, follow it down with the hands. Then hold what you have gained, and with the next full expiration you may be able to get in still deeper, until after a series of deep breaths the desired spot is reached. Naturally this cannot be done if there is much tenderness, but pure nervous spasm may sometimes be overcome in this way.

To make use of the relaxation secured by a hot bath, we need an unusually long tub, so that the patient can lie almost flat when his knees are *slightly* drawn up. If he is doubled up with his knees and head in close proximity, nothing can be accomplished. The patient gets into the tub with the water comfortably warm, and its temperature is then raised to between 110° and 120° F. by pouring in very hot water. The greatest relaxation is usually attained after about ten minutes' immersion. When women are examined the water can be rendered opaque by adding milk or soap suds.

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[1] Special methods of palpating a diseased kidney, spleen, or liver are described in the sections on those organs.

[2] It is essential that the physician as well as the patient should be comfortable during an abdominal examination, else his attention is not wholly on his work. Hence the importance of a high, narrow bed, or table, so that the physician need not stretch or stoop to reach the patient.

This method is far less inconvenient than etherization, and is especially valuable when the recti are well developed and form rounded, tumor like masses as soon as ordinary palpation is attempted. If we suspect that a tumor-like mass may be one of the bellies of the rectus, it is well to grasp the mass with the hand and then ask the patient to raise his head. The mass will harden suddenly if it is the rectus.

2. *What can be Felt Beneath the Normal Abdominal Walls*

No part of the normal intestine, including the appendix, can, in my opinion, be felt through the abdominal walls. The same is true of the stomach, spleen, left kidney, pancreas,[1] bladder, and pelvic organs. All that we can make out in most normal cases is:

1. The abdominal aorta.
2. The spinal column, near and above the umbilicus.
3. Part of the liver (occasionally, if the costal angle is sharp and the belly walls are thin and lax).
4. The tip of the right kidney (in many young persons).
5. Gurgling and splashing in the stomach or colon.
6. The ilio psoas muscle and sometimes the beginning of the iliac arteries in thin people.

The aorta is too deep to be felt at all in some persons, but, on the other hand, it is astonishing how close under the belly wall it is in others, *i.e.*, in those whose dorsal spine projects sharply forward. In such persons the aorta may be almost taken in the hand, and its course, calibre, and motions are so startlingly evident that it is often mistakenly supposed to be the seat of an aneurism, especially as a systolic murmur and thrill can be appreciated over it if a little pressure is exerted, so as to produce an artificial stenosis.

Behind and beside the aorta we can sometimes feel the bodies of the vertebræ, and on them trace the division of the aorta into the common iliacs.

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[1] Leube believes that in very thin subjects the head of the pancreas may occasionally be felt.

The liver cannot be felt at all in the great majority of normal persons, but occasionally the costal angle is so sharp that a small portion of the organ is palpable in the epigastric region.

Bimanually, (see below, page 407) the tip of the normal right kidney may often be caught between the hands at the end of a long inspiration, especially in thin people with lax belly walls.

If the stomach or colon contains fluids, the palpating hand often elicits sounds corresponding to the movement of these fluids. Their only importance in diagnosis will be mentioned later.

Very deceptive often are muscular bundles in the external oblique, which seem distinguishable as sausage-shaped tumors, and doubtless give rise to some of the legends about feeling the normal appendix.

3. *Palpable Lesion of the Belly Walls*

The occurrence of lesions, to be recognized mainly by inspection and percussion, has been discussed (page 355). Besides these we search for:

1. *Herniæ, epigastric or umbilical* (see Fig. 202). The diagnosis rests on the presence of an impulse on coughing, with or without a reducible tumor. Omental herniæ do not bulge with cough.

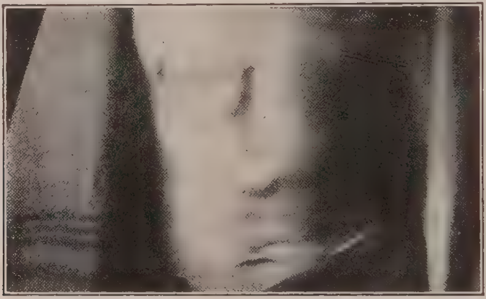

FIG. 202.—Epigastric Hernia.

2. *Separation of the Recti.*—When the patient, lying on the back, lifts his head and shoulders, a longitudinal wedge bulges out along the median line of the belly from the gastric to the suprapubic region.

3. *Abscess of the abdominal walls* usually represents a stitch abscess or the external vent of pus burrowing from the appendix, the pelvis, or the prevesical space. But in about one-third of the cases no such cause can be found. An infected hæmatoma due to trauma or without known cause explains some cases, and occasionally tuberculosis or

actinomycosis occurs. The latter conditions are recognized by the microscopic examination of the pus and of the abscess wall.

4. *Sarcoma* of the belly wall is rather rare, and can be recognized with certainty only by microscopic examination; without this I have known it to be confused with *lipoma* and with *tuberculosis*.

5. *Thickening or inflammation at the navel* occurs in some cases of cancerous or tuberculous peritonitis. The diagnosis rests on the further evidence of cancer or tuberculosis within the peritoneal cavity and on the microscopic examination of a piece excised for the purpose.

Palpation of the Spleen (see page 402).

Palpation of the Liver (see page 378).

Palpation of the Kidney (see page 407).

4. *Study of Abdominal Tumors*

One should notice: *Size*, *contour*, *consistency*, *mobility with pressure and with respiration*, *tenderness*, *pulsation*, *peritoneal crepitus*, *adherence to the skin or to the abdominal wall*, *relationship to any abdominal organ* (also dulness or resonance on percussion.)

Most of these points need no comment. To *ascertain whether the tumor involves the skin*, one lifts up a fold of skin crossing the mass. If the skin dimples markedly over the tumor, *i.e.*, fails to rise at that point while on all sides of the mass it can easily be picked up, the skin is adherent. Tumors in the abdominal wall can usually be gathered up along with the latter when we grasp a large fold with both hands.

To determine the relationship of a tumor with the liver or spleen we note:

(*a*) Whether a groove or interval can be made out, by palpation or percussion, between the mass and either of those organs.

(*b*) Whether its respiratory mobility is as great as theirs.

(*c*) Whether there are other facts in the case suggestive of hepatic or splenic disease (jaundice, ascites, leukæmic blood).

(*d*) The effect of inflation of the colon (see below). Tumors connected with the spleen are forced forward and do not become resonant when the colon is inflated.

To determine the degree of respiratory mobility, hold the fingers of one hand in contact with the lower edge of the mass and allow them to descend with it while the patient takes a full breath. To make sure that an actual descent occurs, one must *sight the mass* (and the hand) against some motionless object in the room beyond, else one may be deceived by the movement of the abdominal walls over the tumor, while the tumor itself remains motionless or nearly so. Tumors connected with the stomach, omentum, liver or spleen move about two inches with a forced inspiration. Kidney tumors move less, seldom as much as an inch. Pancreatic and retroperitoneal tumors have scarcely any mobility. Those connected with the intestine vary considerably in respiratory mobility, according to the presence and degree of adherence to other parts, but their excursion is rarely an inch.

Peritoneal crepitus is a grating, rubbing sensation experienced on light palpation, and due—supposedly—to the presence of a plastic, peritoneal exudate similar to that which produces the friction sounds in pericarditis. Over an enlarged spleen (*e.g.*, in leukæmia) peritoneal crepitus may be due to local perisplenitis, and in perigastritis, perihepatitis, and perienteritis similar crepitus occurs.

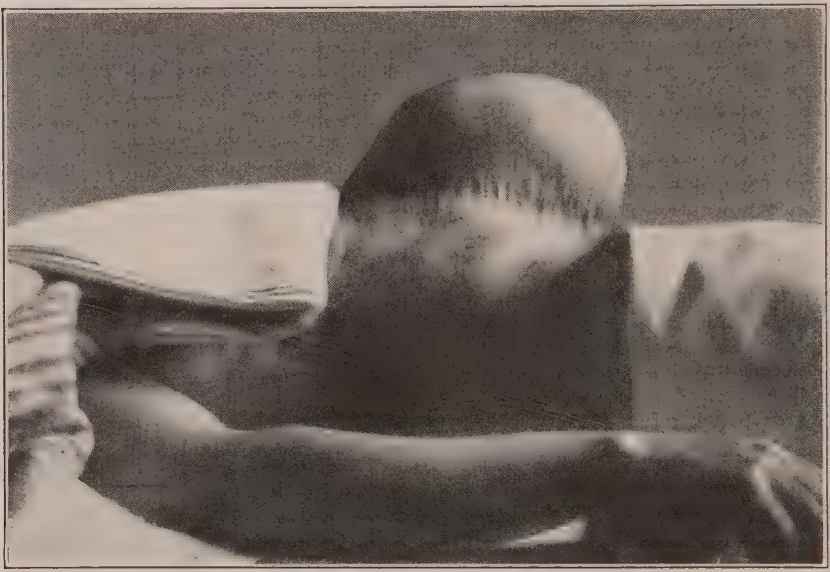

FIG. 203.—Diastasis recti.

Dipping refers to a sudden displacement of the abdominal wall and whatever lies close beneath it, by a swift poke of the finger tips, which may succeed thereby in touching a solid organ or tumor which gentle, gradual palpation misses. Thus one may reach and mark out an enlarged liver through a layer of ascites which would prevent ordinary palpation.

(*a*) *Percussion*

Abdominal percussion is less valuable than thoracic. A lighter blow is used, and the distinction between dulness and tympany is easy. It is of value chiefly to determine the presence of fluid free in the peritoneal cavity, and to ascertain whether a tumor is due to or covered by gaseous distention.

(*a*) *Free fluid* (ascites, peritonitis, hæmoperitoneum, ruptured cyst) gravitates to the flanks and suprapubic region, while the intestines float up and occupy the epigastric and umbilical space. Hence there is dulness in the flanks and over the pubes, with resonance in the epigastric and umbilical regions. But the crucial and ever-necessary test is the shifting of this area of dulness when the patient turns on his side; then the uppermost flank should become resonant and the lower half of the belly—including part of the umbilical region—dull. Without this test the mere marking out of dull areas in the flanks is not conclusive evidence of free fluid there. Occasionally one is deceived by the shifting of a distended colon or a mass of small intestines containing fluid. Still less reliable is the "fluctuation wave," which can be transmitted as an impulse palpable to the hand laid flat on one flank, by sharply snapping the other flank. Similar impulses can be transmitted through the fat of the belly wall, despite all efforts to check them by pressure upon the latter.

(*b*) Percussion is our final test in the diagnostic procedure that begins with *inflation of the colon.* Air is forced into the rectum with an ordinary Davidson syringe, and, as the colon becomes prominent and hyperresonant, we note whether its tympany covers up the tumor-mass under investigation, or whether the mass lies anterior to and remains dull over the inflated colon. Kidney tumors lie behind the inflated colon; splenic tumors remain dull in front of it.

Auscultatory percussion, for identification or demarkation of abdominal tumors and organs, has never been successful in my hands nor in those of most of the observers in whose results I have confidence. Hence I omit further description of it.

Percussion of the stomach and spleen is of very slight value.

Percussion of Traube's space, the "stomach bubble" (the small area bounded on the right by the splenic and on the left by the hepatic dulness, above by the free edge of the left lung, and below by the lower edge of the ribs) is, in my experience, of very little value in diagnosis. This tympanitic area is obliterated in many pleuritic effusions (not in all), but many other causes (full stomach or gut, obese omentum) may produce similar dulness.

Before describing the signs of the different diseases to which the abdominal organs are subject, it seems to me best to introduce here a list of the commoner abdominal tumors found in the study of 4876 such tumors at the Massachusetts General Hospital.

Relative Frequency of Abdominal Tumors

1.	Congested liver	1288
2.	Uterine fibromyoma	766
3.	Hernia	488
4.	Ovarian cyst	382
5.	Gastric cancer	285
6.	Displaced kidney	227
7.	Cirrhotic liver	153
8.	Cancer of liver	151
9.	Cancer of colon	90
10.	Abscess of abdominal wall	79[1]
11.	Splenic tumor in cirrhosis of the liver	60
12.	Leukæmic spleen	58
13.	Malignant tumor of the ovary	43
14.	Tuberculous kidney	41
15.	Tumor as part of tuberculous peritonitis	33
16.	Cancer of the pancreas	32[2]
17.	Neoplasm of the kidney	27
18.	Sarcoma of abdominal wall	27
19.	Enlarged spleen of unknown cause	26
20.	Omental cancer	18
21.	Intussusception	17

II. Diseases of the Peritoneum

1. Peritonitis—local or general.
2. Ascites.
3. Cancer and tuberculosis.

1. *Peritonitis*

1. *Local peritonitis* gives evidence of its presence by (*a*) pain, (*b*) tenderness, (*c*) muscular spasm, (*d*) tumor, and (*e*) constitutional manifestations.

The *pain* may be at first diffuse, later localizing itself at the site of the lesion; or it may be felt first where the peritonitis begins and spread with the lesion if the general peritoneal cavity becomes involved. The character and intensity of the pain vary greatly.

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[1] Some of these were so small as hardly to deserve classification as tumors.

[2] Rarely produces a palpable tumor but is here mentioned for convenience.

Tenderness is the important sign in diagnosis, and helps us to exclude the various colics and other causes of pain which are often relieved by pressure.

Local muscular spasm of the belly muscles to guard the tender lesion beneath is of great value in pointing our attention to the spot affected, though the muscles may be so rigid as to prevent palpation through them. [*Psoas spasm* is described in the section on Appendicitis, see page 392.]

The *tumor* is apt to consist of intestine or other organs matted together by adhesions about the site of the process.

The *constitutional manifestations* are those of *infection*, viz., fever, leucocytosis, anorexia, constipation, often albuminuria and albumosuria.

The commonest causes of local peritonitis are: 1. Appendicitis. 2. Pus tube. 3. Gall-bladder inflammation.

Less common is cancer or ulcer of the stomach or intestine.

2. *General Peritonitis.*—The belly may be generally *swollen* and tympanitic or *retracted* and hard. *General tenderness* is the most important sign. In advanced cases *free fluid in the flanks* may be demonstrated. No sounds are audible as the intestines are paralyzed. Vomiting is the rule, and soon becomes very foul (stercoraceous). There is fever, with a rapid, weak pulse. The mind is clear. The facial expression may be normal. If there is persistent vomiting the face of that condition appears drawn, pinched, anxious, with dark circles under the eyes. The rapid loss of fluid by vomiting accounts for this. *If peritonitis begins in a patient exhausted after operation or chronic disease, there may be no signs unless those of free fluid can be found.*

The leucocyte count is generally elevated, but in the most virulent cases remains normal or subnormal.

2. *Ascites*

The commonest causes are:

(1) Dropsy, from cardiac, pericardial, or renal disease.
(2) Portal stasis, usually from cirrhosis of the liver.
(3) Tuberculous peritonitis.
(4) Cancer of the peritoneum.
(5) Intestinal obstruction.
(6) Acute hepatitis ("yellow atrophy").
(7) Solid ovarian tumors.

The methods of diagnosis of ascites have been explained above. The diagnosis of its cause depends on the history, the results of puncture, and the general physical examination. The contour of the belly is often that pictured in Fig. 204.

3. *Cancer and Tuberculosis of the Peritoneum*

In connection with cancer or tuberculosis of some abdominal or pelvic organ, the disease may become spread throughout the peritoneum with deposits in the omentum and mesentery. The signs are: 1. Tumor masses scattered here and there, sometimes at the navel. 2. Ascites. 3. Emaciation and anæmia.

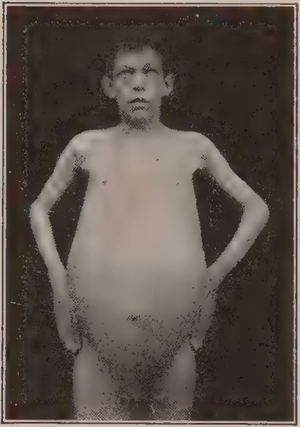

FIG. 204.—Characteristic Shape of Belly in Ascites.

The diagnosis of cancer depends on the recognition of multiple, hard, nodular tumors in the abdomen of a patient known to have cancer of some abdominal organ.

Somewhat similar masses, usually due to loops of intestine matted together by adhesions, may be felt in *tuberculous peritonitis*, but here they are larger, fewer, and not so hard. Cancer appears in late life, tuberculous peritonitis usually in early life. The emaciation and anæmia are less marked in tuberculosis, and fever is more marked. The history or present evidence of tuberculosis elsewhere—lung, pleura, glands, pelvis, testis—favors the diagnosis of tuberculous peritonitis. Cytodiagnosis and the tuberculin test may be of value in diagnosis.

Subphrenic Abscess.—There are two common types—the one near the liver, the other near the spleen.

(*a*) The perihepatic type is recognized, as a rule, chiefly by its etiology (perforated gastric ulcer, appendix-abscess), by the constitutional signs of concealed pus (fever, chills, leucocytosis), and to a less extent by physical signs, none of which, however, serves to distinguish perihepatic from intrahepatic pus. Pain in the hepatic region, prominence of the right lower ribs or right hypochondrium, increased area of percussion dulness over the lower ribs in front and behind (whence empyema or pneumonia is often suspected), and the results of x-ray examination are the data from which we must reason. (See Fig. 198.)

(*b*) The perisplenic type of abscess follows a general peritonitis, which has been treated by drainage and recumbency. The pus becomes pocketed near the spleen instead of gravitating toward the pelvis as it does if the patient's trunk is kept upright.

Pain, sometimes tenderness, the history of the case and the constitutional evidence of concealed pus are the facts on which a conjecture may be hazarded.

The Mesentery

1. *Enlarged glands*—tuberculous, cancerous, or lymphomatous—can occasionally be felt in very thin patients. Their recognition as glands would depend on more obvious evidence of their cause in other parts of the body.

2. *Mesenteric thrombosis* produces all the signs of intestinal obstruction (see below, page 393), from which it can rarely be distinguished without operation, or autopsy, except when it occurs as a complication of mitral stenosis, when we are on the watch for embolism of the brain, extremities and mesentery.

CHAPTER XXII

THE STOMACH, LIVER, AND PANCREAS

I. The Stomach

The best methods of examining the stomach are:

1. Inspection and palpation of the epigastrium and the neighboring portions of the abdomen.
2. Bismuth—x-ray examination.
3. Examination of the stomach contents: (*a*) fasting; (*b*) after a test meal.

By combining the results of these three methods of examination with the results of our general examination of the body—emaciation, anæmia, etc.—and *with the data obtained by a careful history*, we obtain all the information about the stomach which it is possible for us to make use of at the present time.

1. *Inspection and Palpation of the Epigastrium*

(*a*) *Tenderness.*—The normal stomach cannot be seen or felt, nor can anything certain be learned in regard to it by percussion or auscultation. Tenderness in the epigastrium is so common that we can attach no significance to it. In a small portion of cases cutaneous tenderness in the back (lower dorsal or upper lumbar region) can be elicited in cases of peptic ulcer, but this has no value in diagnosis.

(*b*) A *tumor in the epigastrium* (see Fig. 205) is of far greater importance than any other local evidence. If it occurs in an emaciated and anæmic person past middle life, is hard and nodular, and does not disappear after catharsis, it is almost invariably due to cancer of the stomach. Such a tumor may also be due to a mass of adhesions about a gastric ulcer. Tumors of the pancreas much less often reach the surface in this region; tumors of the liver are generally larger, and their connection with this organ can generally be demonstrated by percussion, palpation, and by their greater respiratory mobility when compared with gastric cancer.

Epigastric hernia usually shows an impulse on coughing, is soft and doughy in feel, and presents none of the other symptoms and signs of gastric cancer.

Tubercular deposits in the omentum are almost always associated with ascites, fever, and other evidences of tuberculosis either in the examination of other organs or in the history.

(*c*) *Visible gastric peristalsis* means stenosis of the pylorus (cancer, ulcer, adhesions, or muscular spasm). The contraction wave passes from left to right across the epigastrium, and is seen by means of the shadow cast by a tangential light with the patient in a recumbent position. If the peristalsis stops it can sometimes be reexited by briskly snapping the epigastric region with the finger.

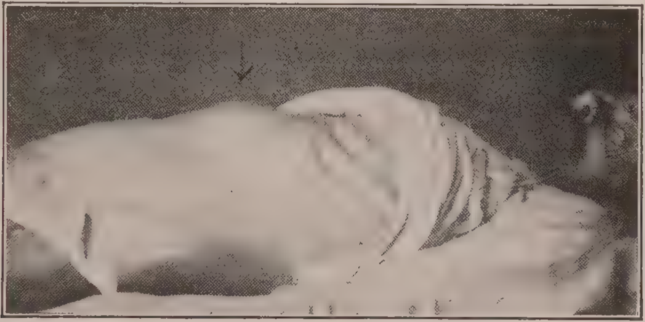

FIG. 205.—Epigastric Tumor in Gastric Cancer.

(*d*) The *normal splash sound* can usually be heard if sudden, quick pressure is made in the epigastrium within three hours after a meal. If splashing can be elicited more than three hours after a meal, and especially if it is present before breakfast, it is evidence of gastric stasis and usually of dilatation.

(*e*) *Hypogastric bulging due to dilated stomach* is occasionally seen in cases of marked dilatation when the patient stands up, and is examined in profile (see Fig. 206).

2. *Use of the Stomach Tube*

(*a*) The *passage of the tube.* The standard red rubber tube generally in use in this country comes in two sizes. Personally I prefer the larger, with a lateral as well as a terminal opening at the lower end, although the smaller size produces somewhat less discomfort. The patient should be covered by a rubber sheet and the clothing removed from the abdomen. So prepared, he should sit in a straight-backed, wooden chair, with a good-sized foot-tub between his feet and a towel in his hand ready to wipe away the profuse secretions of the mouth and pharynx. He should then be warned that the process of passing a tube, although entirely free from danger, is very disagreeable, both on account of the nausea which it produces and because it often seems to the patient as if he were choking and could not get his breath. This, in fact, is not the case, and if the patient will persist in drawing long, deep breaths throughout the process of passing a tube, the worst of it is over in twenty seconds.

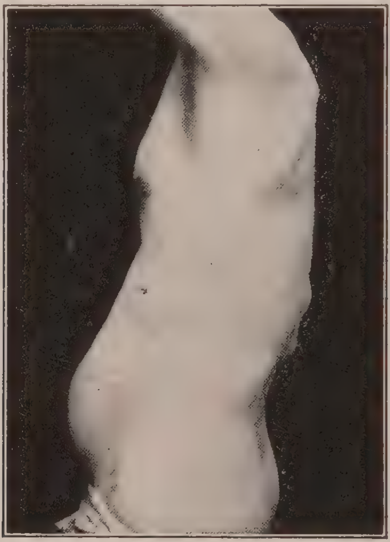

Fig. 206.—Outline of Abdomen in Dilatation of the Stomach, due to cancer at the pylorus.

The tube is moistened with water and pushed straight down through the pharynx without any attempt to direct it, beyond keeping the median line. There is no danger of entering the trachea and no use in trying to avoid it. On its way down the tube is arrested now and then by muscular spasm of the œsophagus, but after a few seconds the spasm relaxes and allows us to push the tube on until the twenty-two-inch mark reaches the teeth. The lower end of the tube is then in the stomach,[1] and we are ready to extract the gastric contents (in case a test meal has been previously given), to wash out the organ, or to distend it with air or water. Since the passage of the stomach tube is the means whereby we become sure of the existence of such diseases as cardio-spasm, diverticulum of the gullet and cancerous stricture of the gullet, some account of the diagnosis of these diseases will be given here.

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[1] Unless there is gastric dilatation or gastroptosis; then the tube must be pushed in several inches farther, the distance depending on the position of the lower gastric border, as determined in previous examinations.

Cardio-spasm with Dilatation of the Œsophagus.—Plummer has reported 40 cases seen in the Mayos' clinic within two and a quarter years. Hence the disease cannot be a rare one though there are surprisingly few cases on record. The patient complains that food sticks, causing discomfort at the lower end of the sternum and later regurgitating unmixed with acid juice. The cases usually begin at so early an age (29 is the average) and are usually chronic enough when seen to exclude a cancerous stricture, but as a rule the first reliable evidence on this point is obtained when we find that:

1. A stomach tube will not pass while a large sound properly guided by a thread (see below) passes fairly easily.

2. Great and long-continuing relief is obtained by dilating the stricture a few times with water pressure inside a silk covered rubber bag.

Radiography of a bismuth meal and the use of the œsophagoscope are supplementary aids to diagnosis. If there is any difficulty in reaching the stomach, a silk thread six yards long is swallowed. After the lower end has passd into the gut the upper end can be pulled taut and on it as a guide a sound and subsequently a dilator can be passed.

Diverticulum of the Œsophagus.—Most diverticula are so high up in the gullet that they are easily recognized by radiography and sounding. The rarer diverticula low down in the œsophagus can also be recognized in most cases by radiographing a bismuth meal or by the silk thread method above described. The thread guides the sound past the opening of the diverticulum.

Cancer of the Œsophagus.—The age of the patient and the duration of symptoms usually differentiate cancer of the gullet from cardio-spasm. In doubtful cases the fact that sounds even when accurately guided do not pass much more easily than the soft rubber tube favors the diagnosis of cancer. *X*-ray settles it.

(*b*) *Extracting the Gastric Contents.*—One hour after a test meal the tube is passed and the patient is then asked to lean forward, press with his hands upon his stomach, and strain down as if he were going to have a movement of the bowels. In most cases this suffices to force the gastric contents out through the tube and into a basin, which is held ready. If the gastric contents cannot be extracted either in this way by having the patient lie down or by moving the tube in the pharynx so as to excite nausea, we should make sure first that the eye of the tube is not plugged. This may be ascertained by disconnecting the funnel and blowing through the tube, which usually suffices to discharge any obstacle from the eye of the tube. If still the gastric contents do not flow out, we may use suction by connecting a Politzer air-bag with the end of the tube in place of the funnel.

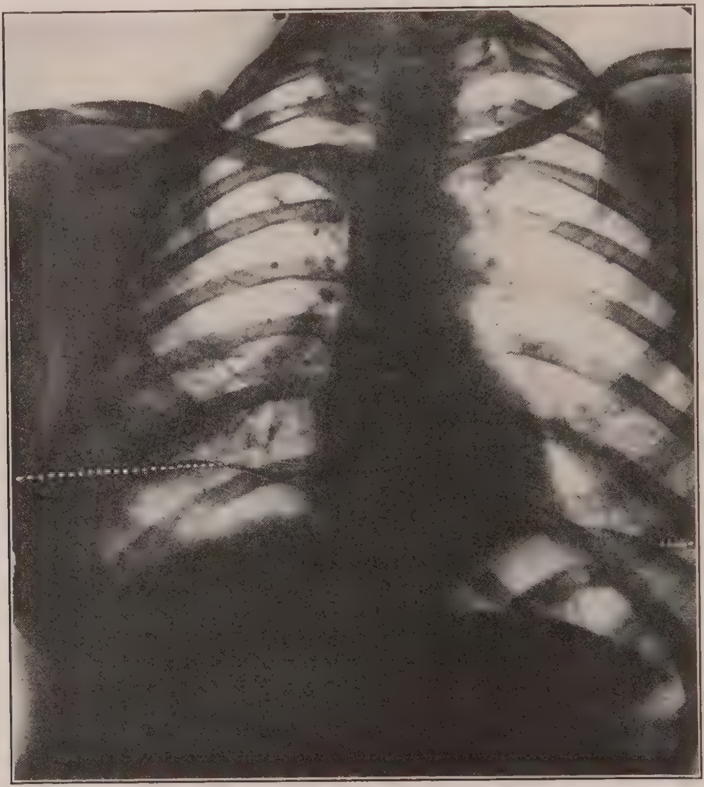

FIG. 207.—Traumatic Pneumoperitoneum from perforation of an esophageal stricture by a sound.

For the analysis of the contents so obtained, see below.

(*c*) *Washing the Stomach* (*Lavage*).—Though not of much use this procedure may be briefly mentioned here. After introducing the tube as above described, about a pint of water is poured in through the funnel, and, just before the water disappears in the vortex of the funnel, the latter is rapidly lowered so as to empty by siphonage into a vessel on the floor. This process is repeated until food and mucus cease to come out and the water runs clear.

To remove the tube at the end of any of the procedures just described, we have only to pinch it tightly just outside of the patient's teeth and pull it rapidly out.

3. *Examination of Gastric Contents*

1. The *contents of the fasting stomach* are best obtained by passing the tube before breakfast, and should consist of no more than a few cubic centimetres of clear fluid containing free hydrochloric acid. If any food is present, gastric stasis is proven. If more than 50 c.c. of fluid without food are present, hypersecretion is indicated.

2. *Gastric Contents after a Test Meal.*—The best test meal is that of Ewald, and consists of a slice of bread (or its equivalent in crackers or cereal) with a glass and a half of water. After this meal not more than 100 c.c. should be found in the stomach at the end of an hour. Occasionally the stomach has emptied itself even within the hour, and we have then to reduce the period.

After extracting the gastric contents as above described and noting the quantity, we should investigate also their *color, odor, and general appearance.* (*a*) Small streaks of blood are of no consequence. Considerable quantities of blood (fresh) suggest ulcer. Small quantities of dark-brown substance resembling blood should be investigated by the guaiac test. If this is positive, gastric cancer is suggested.

The *guaiac test* is best performed as follows: Chip off the oxidized outer shell of a lump of gum guaiac and prepare a fresh tincture by shaking a few chips of the inner non-oxidized guaiac with a few cubic centimetres of alcohol. Add about 10 drops of this tincture and 2 c.c. of hydrogen peroxide to an ethereal solution of the gastric contents prepared by extracting 10 c.c. of gastric contents with 2 c.c. of glacial acetic acid and 15 c.c. of ether (shake 5 minutes). On adding the guaiac to the ethereal solution of gastric contents a *blue color indicates the presence of blood.*

(*b*) For acetic and butyric acids we test merely by our sense of smell. Whenever stasis or fermentation has occurred, we are apt to get a characteristic odor of these acids mingled with that of yeast.

(*c*) The general appearance of the contents tells us little that is important. In cases of marked dilatation they often separate into three layers—the upper frothy, the middle a thin, turbid liquid, and the lower a flocculent sediment of partially digested food.

Mucus is not of any considerable clinical significance unless it is so abundant that the whole stomach contents will slide in one lump from one beaker to another.

When absolutely no digestion has taken place, as in the rare cases of achylia gastrica, the contents consist simply of unaltered bread and water.

(*a*) *Chemical Tests of Gastric Contents*

1. Dip a piece of blue litmus in the contents; if no reddening occurs, no further tests need be made.

2. If the contents are acid to litmus, test with *Günzburg's reagent* (phloroglucin, 2 gm.; vanillin, 1 gm.; alcohol, 30 gm.), by mixing two drops of it with an equal amount of gastric contents (unfiltered) upon a white porcelain plate or dish, and evaporating slowly over a flame.[1] If free HCl is present, a bright rose pink appears. In the absence of free HCl, the color is a dirty yellowish-brown.

If this test is positive, we need make no further tests except the following:

(*b*) *Quantitatiue Estimation of free HCl and of Total Acidity*

To 10 c.c. of unfiltered gastric contents add four drops (about) of Töpfer's reagent (dimethyl-amido-azo-benzol: 0.5 per cent alcoholic solution) in a beaker; a carmine-red color results. Fill a graduated burette with decinormal NaOH solution, and let it run out into the beaker, a few drops at a time, until the carmine-red color disappears. While titrating stir the mixture constantly with a glass rod. Note the number of cubic centimetres of NaOH that have run out.[2]

To estimate the quantity of free HCl, multiply the number of cubic centimetres of NaOH used in the titration by 0.0365; the result is the percentage of free HCl. Normal free HCl varies from 0.07 to 0.2 per cent, or from 2 to 6 c.c. of decinormal NaOH for 10 c.c. of gastric contents ("gastric acidity = 20 to 60").

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[1] The same test may be performed on a glass slide which is subsequently put upon a piece of white paper to bring out the color.

[2] An ordinary medicine-dropper may be substituted for the burette if we get an apothecary to mark with a file upon it the point to which a (previously measured) cubic centimetre of water rises when sucked into the dropper. The half-centimetre point can be similarly marked. Decinormal NaOH solution is then sucked into the dropper and expelled, one-half centimetre at a time, into the beaker containing the Töpfer's reagent and gastric contents.

The estimation of combined HCl and of the acid salts is seldom of importance.

Total acidity is determined by adding to the same beaker of contents in which the free HCl has just been neutralized two or three drops of a one-per-cent solution (alcoholic) of phenolphthalein, and continuing the titration with the NaOH solution (and constant stirring) until a *permanent* red color appears. By multiplying the number of cubic centimetres of NaOH used from the beginning of the first titration up to the point when the red color reappears by 0.0365, we obtain a figure 1 representing the percentage of total acidity. The normal range of total acidity is from 0.15 to 0.3 per cent, and we usually find that we use from 4 to 8 c.c. of NaOH solution in the process of neutralizing 10 c.c. of gastric contents ("gastric acidity 40 to 80").

Lactic acid is to be tested for *only when HCl is absent.* The test must be made at once, since lactic acid soon develops in stomach contents which are kept in a warm place. To perform the test, we dilute a solution of FeCl (strong aqueous) with water until a faint yellow color barely remains. Then fill the concavities of two test tubes with this solution, using one for comparison. If, on adding a few drops of stomach contents to the other, a considerable itensification of the yellow color occurs, lactic acid is almost certainly present. A negative test rules out lactic acid.

The *sediment* need not be examined. It is true that sarcinæ and various bacteria (Boas-Oppler bacillus and others) are often found in cases of gastric stasis, but they add little if anything to the other evidence of stasis more easily obtained—*i.e.*, the symptoms mentioned on page 359, the presence of splashing more than four hours after a meal, the evidence of dilatation or gastroptosis as given above, and the finding of organic acids.

In my own practice I have nearly discarded test meals and chemical analysis. History, physical examination and *x*-ray are usually sufficient for all the diagnosis that we can use. The minor variations in the chemistry of the stomach are not in my opinion significant. Still I am not prepared yet to omit all description of these methods.

4. *X-ray Examination of the Stomach*

From two to four ounces of barium sulphate suspended in milk or gruel are taken on an empty stomach (say at 5 A. M.). Six hours later the patient is radiographed in the upright position. There should be then no bismuth residue in the stomach. The presence of any such residue is strong evidence that stasis and, therefore, some of the causes of stasis,—gastric cancer, peptic ulcer (gastric or duodenal), adhesions or ptosis, are present.

Immediately after this test for stasis, a second bismuth meal is given and the patient is then fluoroscoped at frequent intervals thereafter in search of departures from the normal shape assumed by the bismuth shadow under these conditions and corresponding to the outline of the stomach's interior during peristalsis. If the patient is fluoroscoped lying down, great care should be taken to avoid pressure upon the stomach through the abdominal wall. By such pressure the gastric shadow may be so deformed as to simulate hour-glass stomach and other abnormalities. The tube is to be focussed in all cases upon the third lumbar vertebra, and neither this focus nor the patient's position must change; great distortion of the picture and many false inferences result from failure to follow these rules.

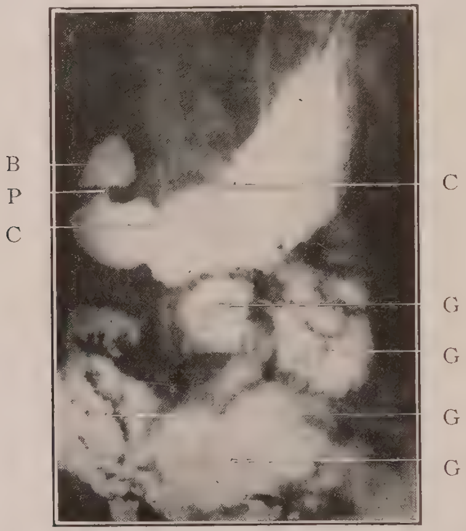

FIG. 208a.—Radiograph of Normal Stomach After Bismuth. B. First Portion of Duodenum. P. Pyloric Ring. C, C. Normal Contraction Rings. G, G, G, G. Bismuth in Gut.

In a satisfactory picture of the stomach one should be able to make out the unbroken outline of the organ indented only by one or two contraction waves. The pyloric sphincter and portion of bismuth just beyond it in the duodenum (the "Bishop's cap") should be visible (see Fig. 208a). If these are not to be made out, or if there is a marked interruption of the normal outline of the stomach shown in the same place in all the plates taken, cancer or ulcer may be suspected (see Fig. 208b).

The changes to be found in peptic ulcer are hyperperistalsis, a filling defect or irregularity in outline and a bismuth residue visible at the end of six hours. These facts, together with the history and physical examination suffice for diagnosis in almost every case.

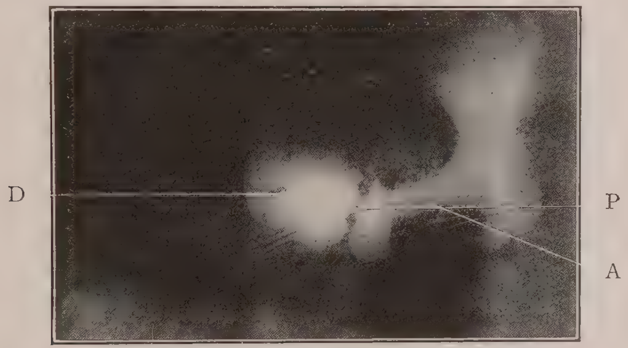

Fig. 208b.—Radiograph of Stomach After Bismuth-Meal. P. Pylorus. A. Loss of outline proved at operation to be due to cancer of the greater curvature and anterior wall. D. Duodenum.

5. *Incidence and Diagnosis of Gastric Diseases*

In the wards of the Massachusetts General Hospital the number of cases apparently of gastric disease treated between 1870 and 1905 was as follows:

Cancer	403
Ulcer	536
Dilatation, cause not determined	170
Dyspepsia[1]	1,002
Total	2,111

The data at our disposal are as follows:

1. The history (most important of all).
2. The local and external examination of the epigastric region.
3. The examination of the gastric contents.
4. The *x*-ray examination.

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[1] *I.e.*, cases of painful digestion including anomalies of motion, sensation, secretion "gastritis" and "gastric catarrh," but without evidence of ulcer or cancer.

(*a*) *In advanced cancer of the stomach* we have pain, emaciation, anæmia, symptoms of fermentation (see page 371), often dilatation and motor insufficiency due to pyloric stenosis, sometimes absence of HCl in the gastric contents (only eighty out of six hundred and fifty cases reported from the Mayos' clinic by Graham and Guthrie showed no HCl), and in about two-thirds of the cases the presence of digested blood ("coffee grounds") in the gastric contents and occult blood (guaiac) in the fæces. *X*-ray usually shows stasis and a defect in the normal outlines. But without the presence of an *epigastric tumor* all these facts are insufficient for diagnosis. Even the tumor itself may deceive us, as the adhesions around a gastric ulcer may present a similar mass to the palpating hand.

The age of the patient is of great importance, especially if during the earlier decades of life he has been totally free from gastric symptoms. *Any type of dyspepsia, any sort of genuine gastric trouble*,[1] *occurring in a person over forty who has never had any such trouble before, is strongly suggestive of cancer.*

(*b*) *Peptic Ulcer, gastric* or *duodenal*.—Physical examination usually shows us very little. *The diagnosis rests upon the history.* Contrary to the usual belief HCl is normal or subnormal in nearly three-fourths of the cases. Occult blood is occasionally found and the stomach may show stasis. The vomiting of blood is infrequent (about twenty-five per cent.). *X-ray evidence is very helpful.*

(*c*) *Pure functional hyperacidity* is not common but may produce symptoms indistinguishable from those of ulcer.

(*d*) *Hypoacidity and achylia gastrica* are not characteristic of any gastric disease. They are common in alcoholism, in all types of anæmia, in tuberculosis, diabetes, and nephritis, as well as in gastric cancer.

(*e*) *Gastric dilatation*, when considerable, is almost always secondary to pyloric obstruction (due to cancer, ulcer, or adhesions). Symptoms suggesting it are the vomiting at one time of a large quantity—a quart or more—of stomach contents, often containing fragments of food eaten more than eight hours previously. Such attacks of vomiting occur usually not after every meal, but at longer intervals. It is to be positively diagnosed by passing a tube and distending the stomach with air or water.

(*f*) *Gastric stasis occurs* in all gastric lesions and in many general diseases (tuberculosis, anæmia, general debility). It constitutes what is referred to by patients as "dyspepsia," or "sour stomach."

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[1] We must exclude angina pectoris and nephritis as well as gall stones and their effects.

Fermentation of contents too long retained is the essential. This results in a sense of epigastric pressure, eructations (gas, sour or burning fluids), anorexia, nausea, and vomiting. Headache, constipation and depression of spirits often accompany it.

(*g*) *Nervous* (*or better emotional*) *dyspepsia* is the commonest of all gastric complaints. Diagnosis rests on the history, the negative physical examination and the results of therapy.

II. The Liver

The Massachusetts General Hospital records (1870–1905) show the following figures bearing on the incidence of diseases of the liver:

Passive congestion	1,288
Portal cirrhosis	234
Biliary cirrhosis (Hanot's)	0
Cancer of the liver	184
Sarcoma of the liver	2
Abscess of the liver	51
Leukæmic infiltration	46
Pseudoleukæmic infiltration	10
Amyloid infiltration	9
Fatty infiltration	6
Hydatid cyst	8
Syphilis	8
"Simple cyst"	6
Actinomycosis	3
Acute yellow atrophy	2
Tuberculosis	1
Total	1,858
Cholelithiasis	457
Acute cholecystitis	110
Catarrhal jaundice	125
Cancer of gall-bladder or ducts	47
Cholangitis	9
Total	701

The evidences of liver disease may be either local or general.

Local signs include: (*a*) Pain and tenderness in the hepatic region. (*b*) Enlargement of the organ, symmetrical or irregular. (*c*) Atrophy of the organ.

The *general signs* which assist in the diagnosis of liver disease are: (*d*) Portal obstruction. (*e*) Jaundice, including changes in the color of the skin, mucous membranes, and excretions. (*f*) Loss of flesh and strength. (*g*) Evidences of infection (fever, leucocytosis, chills, sweats, anorexia). (*h*) Cerebral symptoms (headache, vomiting, depression, delirium, convulsions, coma).

The various attempts to test the liver functions by chemical examination of urine and fæces have not as yet been successful; hence all diagnosis of liver disease must be built up of the above eight groups of data.

1. *Hepatic Pain*

This forms little or no part of many cases of liver disease, since it *occurs only when the capsule is stretched or its nerves are involved in a perihepatitis.* Many cases of hepatic abscess, for example, run their course without pain or become painful only when the pus burrows to the surface and stretches the capsule. Besides this *capsule pain* in liver disease, we have *shoulder pain* referred to the right shoulder or to the region of the right scapula, less often to other parts of the back. Capsule pain is most noticeable in congestion and in cancer of the liver, shoulder pain in abscess.

Tenderness is present in the same cases which are painful, *i.e.*, those in which there is perihepatitis or stretching of the capsule by rapidly increasing tension from within. The latter condition is commonest in passive congestion, but is not characteristic of any single disease.

2. *Enlargement of the Liver*

Tumors behind the liver, pushing it forward and down, are often overlooked, because they bring the liver so prominently into the foreground and fasten our attention on what is mistaken for an enlargement of the organ. Wherever the cause of a supposed enlargement of the liver is not obvious, retroperitoneal sarcoma or some other deep-seated tumor should be suspected.

I have already alluded to the possibility of mistaking the enlarged liver for empyema, and vice versa.

We are sure of an increase in the size of the liver only when we can feel its edge below the ribs and can determine by percussion that its upper border is not depressed.[1] To feel the edge of the liver, hook the fingers of both hands around the margin of the right ribs and ask the patient to take a deep breath. At the height of inspiration an edge may be felt to descend against the fingers and to push its way beneath them. Unless an edge, either sharp or rounded, is felt, one cannot be sure of hepatic enlargement, for percussion of the lower edge of the liver is notoriously unreliable. Dulness below the costal margin is frequently found in cases without hepatic enlargement, and should never be relied on unless the liver can be *felt*.

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[1] A normal liver may be pushed down by air, water, or solid tumors in the lung and pleura, so as to be palpable below the ribs; but the evidence of a cause and the low position of the upper border usually make diagnosis easy.

The long, smooth edge of the liver descending one or two inches with full inspiration is rarely mistaken for anything else, but if the edge is irregular and the surface nodular (see below) it may be hard to distinguish liver from stomach or possibly kidney.

If ascites is present, the presence and dimensions of an enlarged liver beneath the fluid can sometimes be made out by *dipping* (see above, page 360). If this is impossible, the ascites may be tapped, after which it is usually easy to feel any enlargement that is present, as the belly walls are very flaccid.

The *causes of hepatic enlargement* (in adults[1]), arranged approximately in the order of frequency, are:

1. Passive congestion (later stages of uncompensated heart disease).
2. Obstructive jaundice (from any cause).
3. Cirrhosis.
4. Fatty liver, including, "infiltration" and "degeneration."
5. Malignant disease.
6. Syphilis of the liver (congenital or acquired).
7. Abscess of the liver.
8. Leukæmia and pseudoleukæmia.
9. Cholangitis.
10. Amyloid.
11. Hydatid cysts.

The *largest livers* are found in *malignant disease*, *biliary cirrhosis, and abscess*.

In *passive congestion* the liver is very tender, and the presence of uncompensated heart disease[2] usually makes the diagnosis easy. The surface of the organ is smooth and firm.

In *cirrhosis* a distinction must be drawn between (*a*) *latent* or *compensated cases*, wholly without symptoms, and (*b*) *uncompensated cases*, in which diagnosis depends on the chronic enlargement without any considerable increase under observation, associated with evidence of *portal* or *biliary obstruction* (*or both*) and without much pain or irregularity of the liver. Eighty per cent of the two hundred and thirty-four cases recorded at the Massachusetts General Hospital showed enlargement, but only twelve per cent showed pain (*cf.* Malignant Disease, below).

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[1] In infants, rickets, anæmia, and gastro-intestinal disturbances often produce hepatic enlargement, though the splenic enlargement is usually much greater.

[2] Either primary or resulting from arterial disease and hypertension.

The *fatty liver* is soft and smooth in feel. The presence of phthisis or alcoholism makes us suspect this diagnosis, which depends largely on excluding other causes of enlargement.

Malignant disease of the liver (cancer or sarcoma) is usually secondary to new growth elsewhere. The liver *grows rapidly under observation*, is usually *painful* (80 per cent of 168 Massachusetts Hospital cases) and *nodular*. Jaundice and irregular fever are present in over one-half of the cases (54 and 62 per cent respectively), and the loss of flesh and strength is marked.

Obstructive jaundice (due to stone, catarrh, or tumor of the bile ducts, but rarely to any other cause) often produces an enlarged liver. Diagnosis depends on the evidence of a cause for the obstruction and the absence of hepatic nodules, pain, or a rapid increase in the size of the organ.

Syphilitic liver may be distinguishable from cirrhosis or from malignant disease only by the Wassermann test and therapeutic test. The history or present evidences of alcoholism or of syphilis are important factors in diagnosis, but, since syphilis may simulate the nodular liver of malignant disease or the general enlargement and portal stasis of cirrhosis, *it is essential to give antisyphilitic treatment in all doubtful cases of liver disease.*

Abscess of the liver produces enlargement, fever, leucocytosis, and chills in typical cases, but any of these symptoms may be absent and diagnosis is often difficult. Pain is usually absent. The presence of a possible cause (amœbic dysentery, appendicitis) is very important evidence. The *enlargement* is more apt to be *upward and to the right* than in other liver diseases, since the pus usually starts in the right lobe and burrows upward. Hence many cases are mistaken for empyema (see above, page 343). Should swelling or fluctuation appear externally the diagnosis is usually obvious, but in most cases this does not occur. Whenever fever, leucocytosis and dulness in the right lower back appear after an appendix operation with drainage, after a dysentery, or after long continued biliary obstruction (gall stone), hepatic abscess should be suspected. As a rule the diagnosis is made on the etiology rather than on physical signs.

Soft new growths and syphilis may be almost indistinguishable from abscess by local signs, but jaundice is much commoner in malignant disease and the liver of syphilis is often irregular. The history is of value.

Suppurative cholangitis, *subphrenic abscess*, and *pylephlebitis* give us practically the same symptoms as hepatic abscess.

Amyloid liver is recognized by the presence of an appropriate *cause* (chronic suppuration or syphilis) and the evidence of amyloid in other organs (enlarged spleen, albuminuria, diarrhœa). The liver is smooth, not irregular as in hepatic syphilis and cancer.

The *leukæmic liver* is recognized by blood examination; the pseudoleukæmic liver by the normal blood and the histological examination of the glandular enlargements which always accompany it.

Hydatid cyst is rarely to be diagnosed by physical signs. The history of a residence in Australia, Iceland, certain parts of Germany or of the British Isles, is important evidence, since the disease has never been known to originate in North America. Physical examination may enable us to make out that the heptaic enlargement is due to a cystic tumor, tense and elastic, with notable absence of constitutional disturbances (Rolleston) and often an eosinophilia.

3. *Atrophy of the Liver*

Diminution in the size of the liver cannot often be demonstrated satisfactorily during life, since we must rely upon percussion for our evidence, and percussion of the upper and of the lower border of the liver may be rendered difficult by distention of the lung (emphysema) or of the colon. Atrophy may be recognized in a small proportion of the cases of *hepatic cirrhosis* and in *acute yellow atrophy*, but is rarely recognized in either condition. The rapidly fatal course of the latter disease with jaundice and a "typhoidal state" contrasts with the prolonged portal stasis characteristic of cirrhosis.

4. *Portal Obstruction*

A characteristic group of signs manifest the presence of an obstacle to the flow of blood through the portal system. This group includes:

1. Hæmatemesis and dyspepsia.
2. Ascites[1] (see page 361).
3. Splenic enlargement.[1]

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[1] Ascites and splenic enlargement are not purely mechanical phenomena. Toxæmia and sometimes chronic peritonitis or cardiac failure contribute.

4. Collateral dilatation of the abdominal veins (rarely seen in life).

Hæmatemesis is usually due to rupture of dilated œsophageal veins, occasionally to gastritis.

Splenic enlargement is more marked in the rare cases associated with chronic jaundice (*biliary cirrhosis*) and without ascites.

The *cause* of portal obstruction is: 1. Cirrhosis, in ninety-five per cent of the cases. The remaining five per cent is made up of: 2. Acute hepatitis and portal compression by thrombosis or tumors.

5. *Jaundice*

The *yellow staining* of sclera, skin, and mucous membranes, with or without changes in the color of the urine and fæces, is known as jaundice. I have classed it as a general rather than a local sign of liver disease, because it may occur from toxæmia and independent of any lesion of the liver; for instance, in septicæmia, malaria, yellow fever, and pernicious anæmia. It is true, nevertheless, that all jaundice is due ultimately to obstruction in the path of the bile stream. In the toxæmic cases the obstruction is due to inflammation of some of the *small ducts* within the liver. In the cases due to stone or cancer the obstruction is in the *larger bile ducts*, usually the common duct.

Causes of Jaundice.—The four types most often seen are:

1. Jaundice of the new-born (occurs in from thirty to eighty per cent of all children).
2. Catarrh of the bile ducts ("catarrhal jaundice").
3. Gall stones, especially in the common duct.
4. Cancer (pancreas, glands, liver, gall bladder, or bile ducts).

Less common are the cases due to

5. Acute yellow atrophy.
6. Syphilis of the liver.
7. Infectious disease or toxæmia.

Rare causes are:

8. Cirrhosis of the liver.
9. Weil's disease and other types of infectious jaundice.
10. Congenital obliteration of the bile ducts.
11. Family hemolytic jaundice.

The *results of jaundice* upon the body are chiefly the following: (*a*) Slow pulse (often below 60). (*b*) Itching of the skin. (*c*) Mental depression. (*d*) Hemorrhagic tendency (which renders operation dangerous).

In mild cases there is no bile in the urine; in severe cases it is almost always present. The stools are *gray* or *clay-colored* when the obstruction is in the larger bile ducts outside the liver, but in the toxæmic forms of jaundice abundance of bile passses into the intestine and the stools are of normal color.

Diagnosis of the cause of jaundice depends on the following considerations:

1. If it occurs during the first four days of life without any other symptom and passes off within a few weeks, we call it simple *jaundice of the new-born.*

2. If the attack is preceded by gastro-intestinal disturbances, usually in a young person, if pain and hepatic enlargement are slight or absent, and if the jaundice passes off within six weeks, we term it "*catarrhal jaundice*" (though the pathology of this and of the preceding condition is unknown).

3. If there have been attacks of biliary colic (see below, page 385), intermittent fever with intervals of good health, and no considerable or progressive enlargement of the liver or gall bladder, *stone in the common duct* is probably the diagnosis.

4. *Cancer* of the pancreas, duodenal papilla, gall bladder, bile ducts, or of the glands at the hilus of the liver, produces enlargement of the gall bladder, and a jaundice usually painless but of the intensest type known. Loss of flesh and strength is rapid. Cancer of the liver itself gives a rapidly enlarging, nodular liver often with pain, and, in fifty per cent of cases, jaundice.

5. In ordinary *portal cirrhosis*, the rarely occurring jaundice is less intense and permanent, portal stasis is usually evident, and there is generally an enlargement of the liver.

6. Enlargement of the liver with jaundice *lasting for years* in young people is probably due to *biliary cirrhosis*, or family hemolytic jaundice. In some of the cases of this group the red cells can be shown to possess an exaggerated vulnerability, and the blood serum may have unusual autohemolytic powers. Several such cases may occur in a single family.

7. *Hepatic syphilis* produces jaundice in a small percentage of cases, and under these conditions is so apt to be mistaken for cancer that I think in all cases supposed to be cancer in or near the liver a Wassermann reaction should be tried and a course of antisyphilitic treatment given. Other lesions or symptoms of syphilis will naturally influence us.

8. The jaundice secondary to septicæmia, yellow fever, malaria, and pernicious anæmia is usually slight and rarely shows in the urine or bleaches the stools. The evidence of anæmia or of an infection makes clear the nature of the jaundice.

9. Acute yellow atrophy, when in addition to jaundice and a small liver, we have rapid failure of strength and somnolence and other mental disturbance leading to death. The urine is rarely characteristic.

10. Weil's disease is a term sometimes applied to the acute infections of spirochetal origin which are accompanied by jaundice. From catarrhal jaundice it is to be distinguished during life only by convincing evidence of general infection.

6. *Loss of Flesh and Strength*

in cases presenting other signs of liver disease is commonest in uncompensated cirrhosis and in malignant disease, but may occur in gallstone disease, syphilis, or abscess. I have known a physician greatly alarmed at his own rapid emaciation, though his symptoms (jaundice and colic) pointed to stone in the common duct and operation proved this diagnosis correct.

7. *The Infection Group of Symptoms*

These symptoms—viz., fever, chills, sweats, leucocytosis, disturbances of digestion and sleep—are oftenest seen in: 1. Cholangitis. 2. Hepatic abscess.[1] 3. "Ball-valve" or "floating" stone in the common duct. In the last disease jaundice is usually present; in the others usually absent. In cancer of the liver, fever and leucocytosis are often present, but the other signs of infection are rarely seen.

8. *The Cerebral Symptoms of Liver Disease*

These vary from simple depression and apathy to delirium, convulsions, and coma. Severe symptoms are oftenest seen at the end of uncompensated cirrhotic cases; eighty-two per cent of our fatal cases showed during the last days of life symptoms indistinguishable from uræmia. Identical disturbance occurs in acute yellow atrophy.

III. THE GALL BLADDER AND BILE DUCTS

(*a*) *Biliary colic*, and (*b*) *enlarged gall bladder*, with or without *tenderness* and pain, are the data on which (with the evidence of local or general infection, cachexia, intestinal obstruction, and jaundice) our knowledge of gall-bladder disease is built up. In some cases puzzling digestive symptoms closely resembling those of duodenal ulcer are present.

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[1] With or without pylephlebitis.

1. *Differential Diagnosis of Biliary Colic*

Biliary colic, due to impaction of a gall stone in the cystic or common duct, is a sudden pain in the gastric or hepatic region, radiating thence in all directions, but especially to the right shoulder, scapula or back, with fever, chills, and vomiting. In most cases the attack lasts from three to twelve hours (Rolleston) unless relieved by morphine. The pains may be of any degree of severity, and are often accompanied and followed by tenderness over the hepatic region and right hypochondrium. The liver or gall bladder is seldom palpable. Jaundice precedes or follows the attack in about one-half of the cases; in about fifty per cent the stone can be visualized by x-ray.

Renal colic differs in that it usually starts over the kidney (in the back) and radiates down the ureter, while the urine is apt to be bloody but free from bile.

Floating kidney with kinked ureter may produce pains which cannot in themselves be distinguished from biliary colic. The palpation of the floating kidney may be all that makes us suspect that organ to be the cause of suffering.

Peptic ulcer (gastric or duodenal) may produce sharp, paroxysmal pain, but this usually comes several hours after a meal, can be relieved by food, vomiting, lavage, or alkalies, and produces no fever, chill, or sweat. Hyperchlorhydria may produce similar pain at night (the commonest time for biliary colic), but is relieved by food or alkali.

Lead colic is almost always associated with lead dots in the gums and stippling of the red corpuscles (see pages 25 and 467). The history of work as a painter or plumber and the absence of tenderness assist the diagnosis.

Gastric Crises in tabes are often operated on under a false diagnosis of gall stones. Study of the reflexes prevents such a mistake in most cases but lumbar puncture is occasionally necessary for diagnosis.

2. *Enlarged Gall Bladder*

An enlarged gall bladder cannot be felt unless it is *stretched tight* by its contents; a very *tense gall bladder* may be palpable without much enlargement. Probably most enlarged gall bladders are not tense, and so cannot be made out without operation. When palpable the organ presents as a smooth, rounded, pear-shaped tumor at the margin of the ribs in the nipple line.

The causes of enlargement are:

(*a*) *Stone in the cystic duct*, at the neck of the gall bladder.

(*b*) *Cancer of the pancreas* or other tumor obstructing the common duct from without.[1]

(*c*) *Cholecystitis*.

In the first of these jaundice is rarely present (ten to fifteen per cent—Riedel[2]), and colic with or without palpable tumor is our guide to diagnosis.

In cancerous obstruction there is intense and permanent jaundice.

In *cholecystitis* there is usually no jaundice, but all the signs of local and general infection—*pain*, *tenderness*, *leucocytosis*, and *fever*—are present. In acute cases the symptoms, however, may be indistinguishable from those of appendicitis, since the pain may be referred to the navel or even to the appendix region. Many mistakes of diagnosis between appendicitis and acute cholecystitis occur, and must occur until our present diagnostic resources are increased.

3. *Results of Cholecystitis*

(*a*) *Adhesions about the gall bladder* may involve the duodenum or pylorus, and produce kinking and consequent dilatation of the stomach and chronic dyspepsia.

(*b*) *Intestinal obstruction* (see below, page 393) is occasionally produced by the ulceration of a large gall stone from the gall bladder into the intestine, usually the small intestine or duodenum.

IV. THE PANCREAS

Diseases of the pancreas can very rarely be diagnosed by our present methods. If greatly enlarged (tumor, cyst, hemorrhage) it may become palpable as a deep epigastric tumor, but we are rarely able to differentiate such tumors from those of the retroperitoneal structures.

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[1] Courvoisier has shown that if the common duct is obstructed by a gall stone the gall bladder is very rarely enlarged.

[2] Riedel: Berlin. klin. Woch., 1901, No. 3.

Indirect and uncertain information is afforded by the presence in the urine of *sugar or fat-splitting ferments*[1] and in the stools by the appearance of an abnormal amount of *muscle fibre* or of *fat* not otherwise to be accounted for (*i.e.*, in the absence of jaundice, diarrhœa, tuberculous peritonitis, or large meals of fat).

1. *Cancer of the pancreas* may sometimes be suspected on account of its pressure effects. Intense and painless jaundice with enlarged (perhaps palpable) gall bladder and liver is often due to the pressure of cancer in the head of the pancreas upon the common bile duct. Ascites and swelled legs may be produced by compression of the inferior vena cava. But the diagnosis can rarely be more than a suspicion, for cancer of the gall bladder, ducts, duodenal papilla or retroperitoneal sarcoma may produce similar pressure effects. Should these pressure effects coincide with a glycosuria and the presence of a deep-seated, almost immovable tumor, the suggestion of pancreatic disease becomes more plausible.

2. *Acute pancreatic disease*, hemorrhagic or suppurative, is not recognizable until it is seen at an operation undertaken for the relief of some grave, acute lesion of the upper abdomen. Perforated gastric ulcer and intestinal obstruction may give identical symptoms, viz., sudden, intense, epigastric pain and tenderness, with vomiting and collapse. One or two days later a tender epigastric tumor may appear, but this presents no characteristic peculiarities.

3. *Pancreatic cyst* presents a very slow-growing, possibly elastic, deep-seated epigastric tumor, which usually produces little in the way of pressure effects, and may be associated with glycosuria and fatty stools.

4. *Bronzed Diabetes.*—The association of diabetes with bronzing of the skin and enlargement of the liver is strongly suggestive of an associated chronic fibrous pancreatitis.

In any doubtful case the possibility of pancreatic disease is increased: (*a*) If improvement follows the administration of pancreatic preparation; (*b*) if glycosuria follows the administration of 100 gm. of glucose (alimentary glycosuria).

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[1] The suspected urine is neutralized with potassium hydroxide and one portion of it boiled to destroy any ferment that may be present. To this and to the unboiled portion ethyl butyrate is added. In twenty-four hours an acid reaction may appear in the unboiled specimen if it contains a ferment, while the other specimen shows no considerable change in reaction.

(*a*) *Incidence of Pancreatic Disease*

The following table is from the Massachusetts General Hospital records (1870–1905):

	Cases
Cancer of the pancreas	35
Acute pancreatitis	13
Chronic pancreatitis	10
Cyst of the pancreas	3
Total	61

CHAPTER XXIII

THE INTESTINE, SPLEEN, KIDNEY

I. THE INTESTINES

Incidence of Intestinal Disease (excluding diarrhœa and constipation) *at the Massachusetts General Hospital*, 1870–1905.

1. Appendicitis	3,314
2. Acute obstruction	142
3. Cancer (above the rectum)	155
4. Dilated colon	6
5. Tuberculosis	2
6. Fæcal impaction (above the rectum)	2
Total	3,621

1. *Data for Diagnosis*

THE data on which are based all our conclusions regarding intestinal disease are obtained from the following sources:

1. *Pain* (colicky or steady) and *tenderness*, *tenesmus*.

2. *Gaseous distention* and the noises and sensations produced by gas.

3. *Diarrhœa or constipation.*

4. *Muscular rigidity* of the belly wall protecting an intestinal lesion.

5. *Tumor*, palpable or visible, and believed to be connected with the intestines (together with the effects of catharsis on such tumor).

6. *Visible or palpable peristalsis.*

7. *Digital or visual examination of the rectum.*

8. *Examination of the intestinal contents*, fæcal and other.

9. *Inflation of the colon* through the rectum.

10. *X*-ray after barrina enema.

11. *Constitutional manifestations*, such as fevers, vomiting, leucocytosis, emaciation.

Some of these data need further comment.

Intestinal Pain.—Most pains associated with intestinal disease (appendicitis, cancer) are due in fact to irritation of the peritoneum.

Which of the numerous pains referred to the belly should be interpreted as *intestinal* in origin? Those especially which (*a*) shift rapidly from place to place; (*b*) accompany the noises and sensations of the passage of gas and fæces through the intestine; (*c*) accompany diarrhœa or constipation; recur rhythmically.

Tenderness is usually a symptom of peritoneal rather than intestinal irritation. With true intestinal pain (*colic*) there is often *relief by pressure*—the precise opposite of tenderness. Yet so close is the association of intestine and peritoneum that in appendicitis, intestinal ulceration, tumors, and even in simple gaseous distention of the gut, there is often local or general tenderness. When extreme and associated with constitutional manifestations—fever, leucocytosis, collapse—it always suggests peritonitis. When there are no constitutional manifestations, a purely *local* pain or tenderness has little *diagnostic* value.

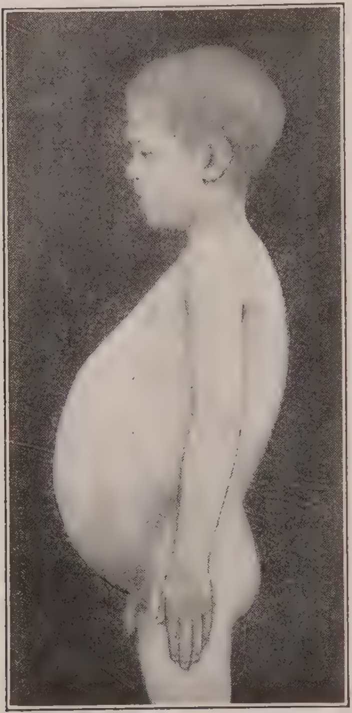

FIG. 209.—Congenital dilatation of colon.

Tenesmus.—The desire to pass another stool as soon as one has been evacuated, together with local burning and straining, means always *rectal* irritation (inflammation, ulcer). It is one of the most definite and reliable symptoms known.

Gaseous distention of the intestine is proved by an *increase of the normal tympanitic* note over part or all of the belly, together with a *prominence* of the overlying belly wall. It is chiefly and most frequently the colon that produces distention.

The *significance of distention* is vague and depends largely on the associated data. In acute *gastro-intestinal "catarrh"* the diarrhoea and absence of severe constitutional manifestations make us put little stress on the associated distention. In *typhoid fever* distention results from atony of the intestinal walls and is "to some extent a measure of the intensity of the local lesions" (Osler). In *intestinal obstruction* distention may be extreme if the stoppage is low down (in the colon), less marked if the lesion is high up.

Distention which continues despite free purgation is very often due to chronic intestinal obstruction.

In *starvation*, children often get very large bellies, owing to muscular atony of the gut and the resulting gaseous accumulation. But in no case is the distention of itself of much diagnostic value. The associated symptoms give it significance.

In *congenital dilation of the colon* (Hirschsprung's disease) a huge belly is associated with obstinate constipation. The colon can be measured and shown to be dilated through the use of bismuth suspensions (by rectum) and *x*-ray. (See Fig. 209.)

Diarrhœa, the passage of more and looser stools than is normal for the individual, is, like distention, a result of many causes both within and outside the intestine.

The most important are:

1. Intestinal disease.
   - (*a*) Indigestion (acute and chronic).
   - (*b*) Ulceration (some cases only).
   - (*c*) Cancer of the colon or rectum.
   - (*d*) Intussusception.
   - (*e*) Infectious diseases (cholera, dysentery, typhoid).
   - (*f*) Intestinal parasites.
2. Outside influences.
   - (*a*) Nervous causes (emotion, Basedow's disease, etc.).
   - (*b*) General infections (sepsis).
   - (*c*) Cachectic states (anæmias, nephritis, etc.).

By a search for these causes, as well as by the use of the data obtained by examination of the stools, we arrive at an understanding of the diagnostic significance of diarrhœa.

Aside from symptomatic diarrhœa, constipation, and dysentery, which produce no physical signs beyond those described—distention, borborygmi, pain, tenderness, tenesmus, and constitutional manifestations—there are but three important[1] diseases of the intestines:

I. Appendicitis.
II. Intestinal obstruction.
III. Cancer of the bowel.

2. *Acute Appendicitis*

1. The local signs are pain, tenderness, muscular spasm, and tumor.

2. The general or constitutional signs are fever, chill, rapid pulse, vomiting, constipation, frequency or cessation of micturition, and leucocytosis.

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[1] Tuberculous enteritis and pericæcal tuberculosis will be briefly referred to later.

(*a*) The *pain* may be at first epigastric (pylorospasm?) or general, later localizing itself in the right iliac fossa, less often near the navel, the gall bladder, or in any other part of the belly.

(*b*) The *tenderness* is more important in diagnosis; indeed, without tenderness diagnosis is rarely possible. It is usually greatest near a point half-way from the anterior iliac spine to the navel. Occasionally a tender point in the pelvis may be reached by rectal or vaginal examination, but this is not at all a reliable sign.

(*c*) *Muscular spasm* over the appendix region is present in most cases, and, while it renders accurate palpation impossible, it is in itself so characteristic of the disease that we do not regret it.

Psoas spasm occurs in a minority of cases. The patient leans his body forward and toward the right in walking, or, if recumbent, draws up the right thigh to relax the spasm.

(*d*) *Tumor*—about the size and shape of a lemon, ill-defined and tender—is felt in the right iliac fossa in many cases. It may be considerably larger and better defined if abscess has existed for several days, or it may be smaller and more sausage-shaped. Fluctuation and bulging can sometimes be made out by rectum or vagina.

(*e*) The *constitutional signs* may or may not be marked, according to the duration of the process, its virulence, and the degree of infection of the peritoneal cavity. The fever is usually moderate, under 102° F., with corresponding elevation of the pulse. Vomiting comes at the outset if at all, and is usually over by the second day. A leucocyte count which rises or remains elevated (above 16,000) accompanies the active and advancing stages of the disease. In cases that are very mild or tightly walled in by adhesions, and in cases with virulent general peritonitis, the leucocytes may be normal or subnormal.

Diagnosis can hope only to establish the existence of a local inflammatory process in the abdomen; acute cholecystitis and acute pus tube may present signs indistinguishable from those of appendicitis, though the site of tenderness often sets us right. Non-inflammatory processes, such as lead colic, tabes, biliary and renal colic, floating kidney, and acute gastro-intestinal upsets, can usually be excluded, since they do not show so much local tenderness, fever, and leucocytosis.

In those who are familiar with the symptoms of appendicitis, a vivid imagination may conjure up a set of sensations that are difficult for the physician to distinguish from those of the actual disease. Even tenderness may be simulated, but, by distracting the patient's attention while we palpate, we may be able to press hard over the appendix without eliciting complaint. The absence of leucocytosis, the age and sex of the patient, also help us to exclude appendicitis.

3. *Chronic Appendicitis*

A disease which can be diagnosed by the x-rays alone. There are no symptoms or signs characteristic of it and in most cases it represents only a harmless historical landmark, a shrivelled appendix.

4. *Intestinal Obstruction*

(*a*) *Acute Obstruction.*—A person may have had no fæcal discharge for a week or even considerably longer and yet present all the evidences of good health. It is only when *vomiting*, severe paroxysms of *pain*, and *distention* of the belly ensue that we suspect obstruction. In the acute cases *tumor* is noted in only about fifteen per cent. In the chronic cases, usually due to stricture or cancer, a fæcal tumor can often be felt and diarrhœa be the chief symptom or may alternate with constipation.

By physical signs alone I do not believe that general peritonitis and acute intestinal obstruction can always be distinguished. Fever is not distinctive of general peritonitis, for it occurred in eighty-four out of one hundred and twenty-two cases of acute obstruction in the Massachusetts Hospital records, and in forty-three of these cases free fluid in the peritoneal cavity was demonstrated as well. Stercoraceous vomiting may occur in general peritonitis; it was absent in three-fourths of the Massachusetts Hospital cases of obstruction. Weak, rapid pulse, cold extremities, and a drawn, anxious face are common to both diseases. Tenderness is more general and more marked in general peritonitis than in simple obstruction, yet some tenderness was complained of in fifty-six out of the one hundred and twenty-two cases of obstruction just cited.

On the whole, the differential diagnosis of these two diseases seems to depend far more on the history and the etiology than on physical signs.

(*b*) *Chronic Obstruction.*—Here the diagnosis is simpler. There is usually a history of increasing constipation sometimes interrupted by occasional attacks of diarrhœa.[1] *Tumor* is palpable in fifty-eight per cent of cases. *Visible peristalsis* was recorded in seventeen per cent of the Massachusetts Hospital cases. Distention is gradual and late, but often persists or recurs despite purgation. Cancer, usually at the sigmoid or cæcum, is the commonest cause. Stricture, except cancerous stricture, is rare, but syphilis occasionally produces it.

(*c*) *Acute Obstruction by a Chronic Lesion.*—Cancer of the sigmoid often exists for months almost latent, or produces only moderate constipation, so that the patient considers himself well. Such cancers may present an annular growth, hardly bigger than a signet-ring, practically an annular stricture.

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[1] The latter combination occurred in six per cent of the Massachusetts Hospital cases.

This stricture may be suddenly "*shut down*" during an acute gastro-intestinal attack, and we are then confronted with all the signs of acute obstruction. Only the seat of the lesion, the age of the patient, and possibly the appearance of peristaltic waves can lead us aright in our diagnosis of the cause of obstruction.

5. *Cancer of the Bowel*

The signs are those of chronic intestinal obstruction (see last section). Occasionally the tumor may not produce much obstruction, and we have simply pain and a tumor which we find by examination is not attached to the liver, spleen, kidney, or stomach, and usually is about the size of a hen's egg. If fæces have accumulated behind such a tumor, we may feel larger masses. In my experience palpable tumors due to fæcal impaction alone, without organic stricture or cancer, are very rare, except in the rectum; if found above this region they are almost invariably dependent on stricture or cancer of the bowel.

6. *Examination of Intestinal Contents*

1. *Weight.*—With the average diet of the adult "Anglo-Saxon," the weight of the daily stool is from 100 to 250 gm. (about 25 to 70 gm. dry) but Chittenden has shown that with a low proteid diet of 2,000–2,750 calories value, the weight of the stool may be less than half this amount.[1]

2. *Color.*—(*a*) White or light yellow—milk diet, bread and milk diet.

(*b*) Black—blood, bismuth or iron (medicinal), blackberries, huckleberries, red wine.

(*c*) Green; some normal infant's stools after standing; fermented infant's stool if green when passed; green vegetables, calomel.

(*d*) Gray—absence of bile (jaundice), sometimes after cocoa or chocolate.

(*e*) Bloody red—if in small amount and fresh, usually due to hemorrhoids; in large amounts it may also be due to hemorrhoids or to any of the causes of intestinal ulceration (typhoid, cancer, dysentery, etc.).

3. *Odor.*—In adults of no great significance. In infants foul stools suggest albuminoid decomposition, and strongly sour stools suggest acid fermentation.

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[1] "Physiological Economy in Nutrition," 1904, p. 42.

4. *Abnormal Ingredients.*—(*a*) *Undigested food* in small quantities is present in normal stools, but when digestion is faulty larger quantities easily recognized by the naked eye may occur. Pieces of meat, flakes of casein (especially in typhoid patients overfed with milk), fragments of starchy food, and lumps of fat (steatorrhœa) may be seen.

The natural inference from the presence of these substances is that the gastro-intestinal tract is not at present dealing with them satisfactorily. Fatty stools are present in jaundice, tuberculosis, or amyloid of the intestine, and even in simple catarrh. Though often associated with pancreatic disease, fatty stools are by no means characteristic of it.

(*b*) *Mucus.*—Small shreds of mucus adherent to fæces are of no importance and cause much unnecessary worry among anxious mothers. Larger amounts, if intimately mixed with the stool, point to catarrh of the small intestine; if mucus thickly coats or makes up the bulk of the stool, the trouble is in the colon. The latter is by far the commonest condition. Anything from a very mild to a severe catarrhal condition is accompanied by mucus. Large periodic discharges of mucus and shreds mean usually the neurosis "colica mucosa."

(*c*) *Fresh Blood.*—Piles are by far the commonest cause of bloody stools, and the amount of blood may be trifling or may be large enough to produce in time a severe anæmia.

Enteritis (the mild follicular or the severe ulcerative form) often produces bloody stools. The associated symptoms, diarrhœa, mucus, and pain, together with the etiology (dietetic error, typhoid fever, amœba histolytica), must determine the nature of the enteritis.

In *cancer* of the rectum or sigmoid (rarely higher up in the bowel), small quantities of blood, fresh or altered, are almost always passed sooner or later. The infrequent, offensive, and painful stools and the results of digital examination usually reveal the source of the blood.

In *intussusception* the association of bloody stools with the sudden appearance of a painful abdominal tumor (usually in the cæcal region), vomiting, and severe constitutional manifestations suggest the diagnosis.

In *hemorrhagic diseases* (purpura, scurvy, acute leukæmia) blood may come from the intestine as well as from the other mucous membranes. Other rare causes for blood in stools are a ruptured aneurism, thrombosed mesenteric artery, rectal syphilis, or fissure.

(*d*) *Altered blood* (tarry stools, melæna) follows the pouring out of blood—a pint or more—in the upper gastro-intestinal tract, and occurs in hepatic cirrhosis, gastric or duodenal ulcer, after severe nose-bleed, and occasionally from other causes. *Occult blood*, recognizable by the guaiac test, often occurs in cancer or ulcer of the stomach, and forms an important link in the chain of evidence on which the diagnosis of those diseases is based.

(*e*) *Pus* is not of great diagnostic value. Large amounts mean the breaking of an abscess (appendix, pus tube) into the rectum. Small amounts occur in ulcers or even from catarrh.

(*f*) *Shreds* of tissue point to ulceration.

(*g*) *Gall Stones.*—In suspicious cases break up the fæces in a sieve with plenty of water. The peculiar, *facetted* shape of most gall stones is easily recognized.

7. *Intestinal Parasites*

Bacteria.—Only the tubercle bacillus can be recognized without culture methods, which do not fall within the scope of this book.

For the identification of tubercle bacilli the following method is to be recommended: "Dilute the stool with ten volumes of water, mix thoroughly, and let it stand in a wide-mouthed bottle for twenty-four hours. The narrow layer between the thin supernatant liquid and the solid sediment contains the bacilli. Remove this with a pipette, spread it on a cover slip, evaporate slowly to dryness, and proceed as with sputum" ("Harvard Outlines of Medical Diagnosis," 1904, p. 29).

Animal Parasites.—The most important are:

I. Serious....	1. Amœba histolytica.	
	2. Hook-worm	(*a*) Uncinaria americana. (*b*) Anchylostoma duodenale.
	3. Bilharzia hæmatobium.	
II. Relatively mild.	4. Balantidium coli.	
	5. Tape-worms; the beef-worm (Tænia saginata) is very common; the pork-worm (Tænia solium) is rare; the miniature tapeworm (Tænia nana) and the fish-worm (Dibothriocephalus latus[1]) are fairly common. Several other forms occur in foreign countries.	
	6. Strongyloides intestinalis.	
III. Usually harmless.	7. Ascaris lumbricoides (round-worm).	
	8. Oxyuris vermicularis (thread-worm; pin-worm).	
	9. Trichiuris trichiura (whip-worm).	
	10. Trichomonas intestinalis.	
	11. Lamblia intestinalis.	

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[1] Fish tape-worms may produce a severe anæmia, but in probably the great majority of all cases they do not do so.

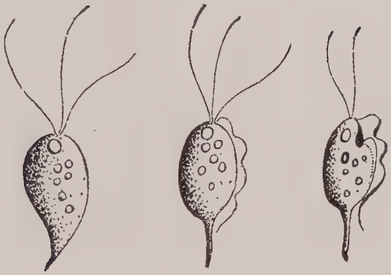


FIG. 1.—Trichomonas hominis. (Leuckart.)

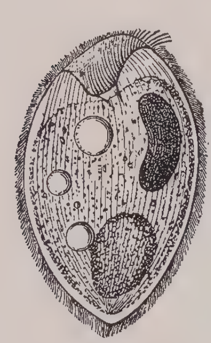


FIG. 2.—Balantidium coli. (Leuckart.) Magnified about 150 diameters.

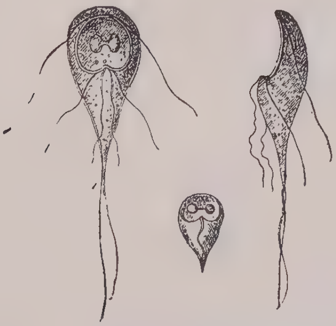


FIG. 3.—Lamblia intestinalis. (Leuckart)

Tape-worms, round-worms, pin-worms, and the strongyloides are to be recognized in their adult form (see Figs. 210, 211, 212, 213, 214). They are usually noticed by the patients themselves and brought to the physician for examination. If the worm has the look of a common earth-worm, but a length of five to nine inches, it is safe to call it the "round-worm" (Ascaris lumbricoides); if the worm is about one-half an inch long and as thick as a pin, it is in all probability a "pin-worm" (Oxyuris vermicularis).

The *Amœba histolytica* is to be searched for in fresh stools passed into a warm vessel, after $MgSO_4$ has been given. A bit of mucus from such stools or a little obtained by passing a rectal tube is put upon a warmed slide with a drop of water, covered with a cover glass, and examined at once with a high-power dry lens. The organism is recognized as an amœba by the presence of *distinct amœboid movements*. When dead it assumes a round shape, but one should not attempt a positive diagnosis until live amœboid parasites are present.

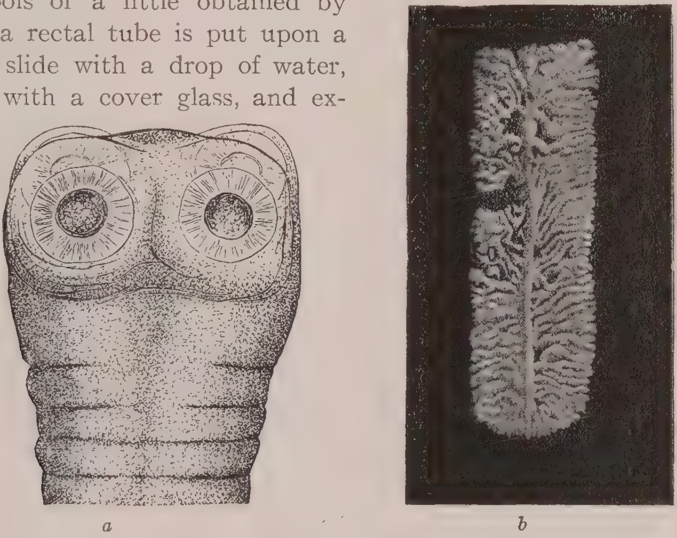


FIG. 210.—*a*, Head of Tænia saginata, much magnified; *b*, uterine canal of same. About twenty branches on each side.

Apparently there is a harmless variety of *amœba coli* to be obtained from the stools of many normal persons by purgation. This is distinguished from the amœba histolytica by the following criteria (Vedder). The dysenteric or tissue-destroying amœba is larger, more actively motile, has an easily distinguished refractive ectoplasm which can also be made out in the pseudopods which are themselves relatively large and easily seen.

Especially characteristic of the *amœba histolytica* is the presence of the numerous *vacuoles* and usually of *ingested red corpuscles* which hide the nucleus.

The other parasites are identified, as a rule, by the finding of their eggs in the stools. The technique of this operation is described below, as exemplified in the search for the egg of uncinaria—at present the egg most important for Americans to recognize.

Eggs of parasites catch the eye in the examination of stools, *first* of all, by the clean-cut, *mathematical symmetry of their oval*, when compared with the irregular, shapeless masses which usually appear in slide and cover preparations from the fæces.

Secondly, the *size* of parasitic eggs is greater than that of most of the objects seen in the fæces; and, *thirdly*, they are for the most part dark brown, *stained with bile* (the uncinaria is an exception).

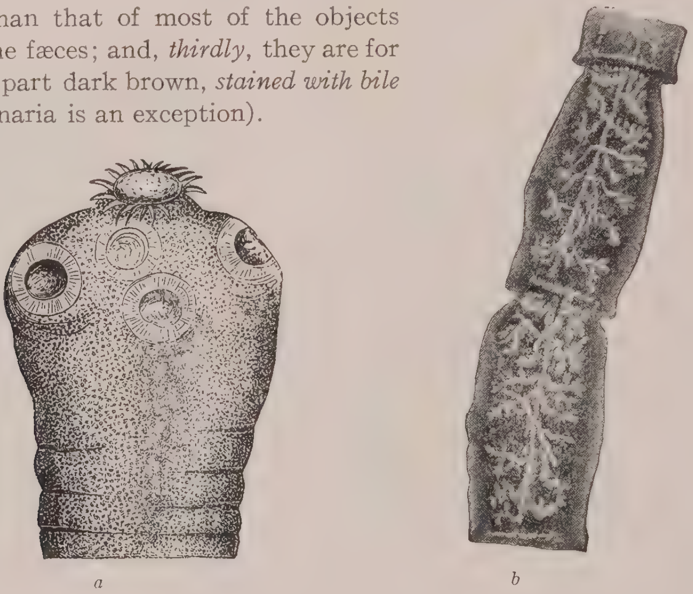
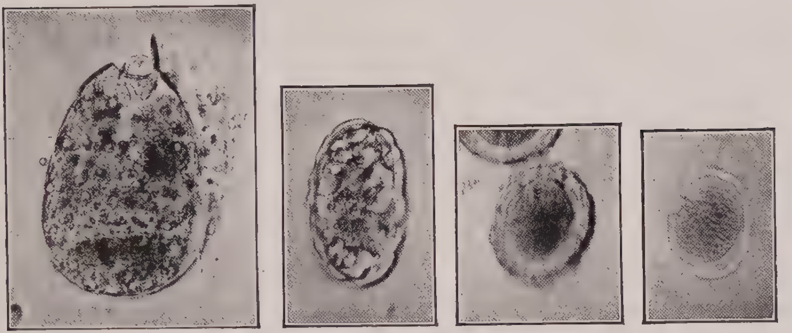


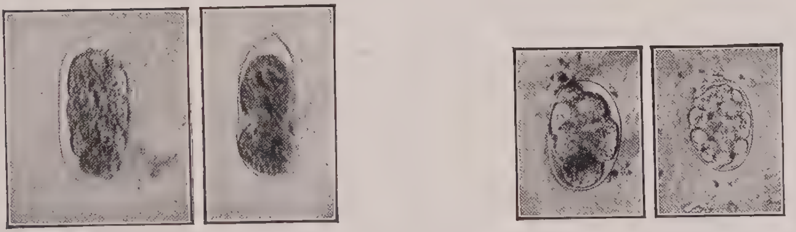
FIG. 211.—*a*, Head of Tænia solium (note crown of hooks); *b*, uterine canal in two segments. Only five to seven branches on each side.

The differences between individual species will be described later. In Plates II. and III. the most important eggs are pictured and catalogued.

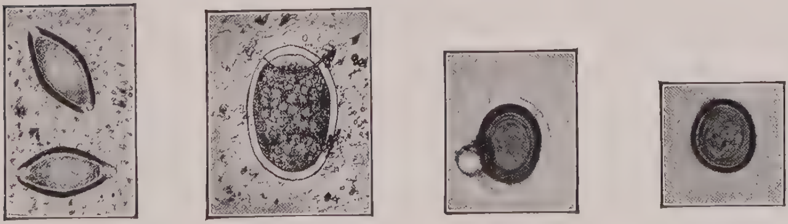
The *Uncinaria americana* or its European equivalent (*Anchylostoma duodenale*) is recognized most easily by the identification of its eggs in the stools. These eggs are characteristic (see Plate II.), and "the only thing liable to be confounded with them is the ovum of Ascaris lumbricoides stripped of its heavy, bile-stained outer shell (see Plate II.); but this has a double contour and contains a shapeless mass of granular matter not differentiated" (as most uncinaria eggs



Distoma buski. Ascaris lumbricoides.



Uncinaria americana. Anchylostoma duodenale.



Trichuris trichiura. Dibothriocephalus latus. Tænia solium. Tænia saginata.

EGGS OF INTESTINAL PARASITES.

All are magnified 250 diameters.

are) "into clear segments."[1] The greater size of the American hookworm's egg compared to that of the European worm is shown in Plate II. "Free embryos are rarely if ever found in intestine. When free (worm-like) embryos are seen in the stools, they are generally those of the Strongyloides intestinalis" (see Fig. 214).

The ova of uncinaria catch the eye in a rapid examination, first, because they are "*not generally bile-stained, but clear*, whereas those of the commonly associated intestinal parasites are of a yellow to deep amber or brown color." They are distributed quite evenly throughout the entire fæcal mass; hence, in searching for them, the following method is advisable:

Technique of Microscopic Examination.—"A bit of fæces the size of a match head is removed with a toothpick and placed on a glass slide. Upon this is placed a cover glass and pressed down so as to give a clear centre to the specimen. Do not add water. Examine with a one-third to two-thirds objective, a No. 4 ocular, and a partially closed

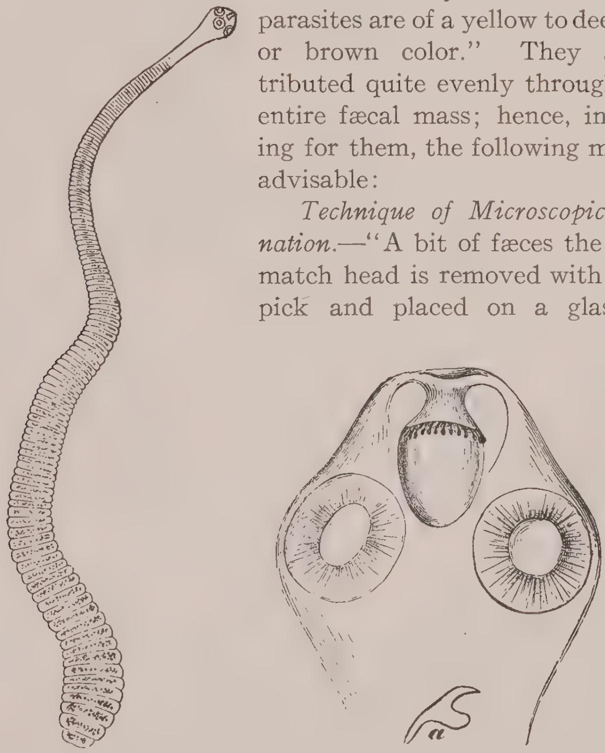


FIG. 212.—Tænia nana (Dwarf Tape-worm). *a*, Hooklet; *b*, head, greatly enlarged; *c*, whole worm, magnified about 10 times.

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[1] All the quotations in this section are from the "Report of the Commission for the Study and Treatment of Anæmia in Porto Rico," by Ashford, King, and Igaravidez (December 1st, 1904), a study of 5,490 cases.

diaphragm. If too much light is admitted the delicate ovum will be passed over."

The following interesting table (from the studies of Ashford, King, and Igaravidez in Porto Rico) shows, roughly, the relative frequency (in a tropical climate) of the common intestinal parasites recognizable by their eggs. In the examination of the stools of 5,490 cases of uncinariasis they found as well:

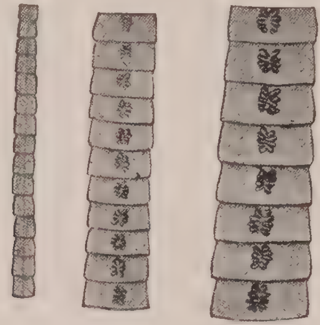


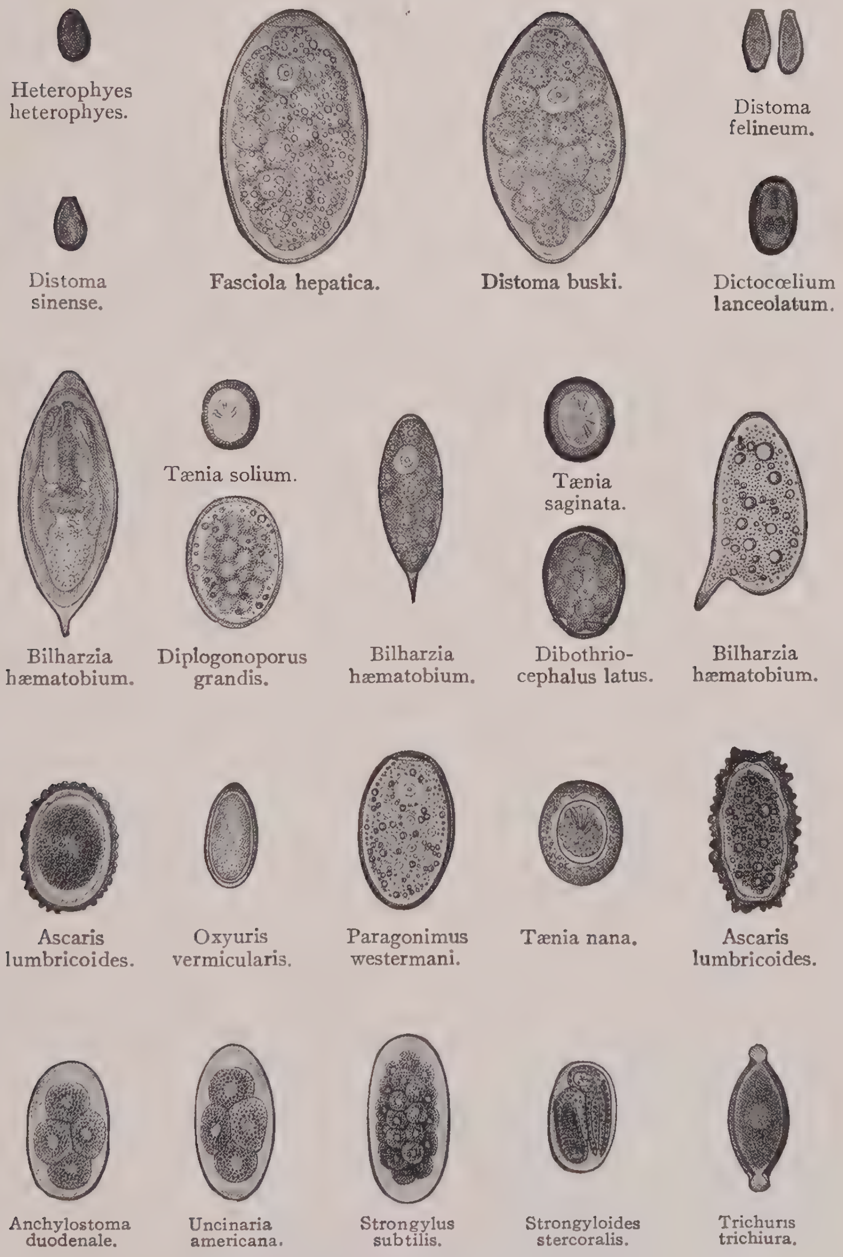
FIG. 213.—Segments of the Dibothriocephalus latus (Fish Tape-worm). Note the rosette-shaped uterine marking.

Ascaris lumbricoides in.......	1,408	(many others seen but not noted).
Trichuris trichiura in.........	326	(many others seen but not noted).
Strongyloides intestinalis in....	36	(the embryo worms, not eggs).
Bilharzia hæmatobium in......	21	(frequently no careful search was made for this egg).
Balantidium coli in...........	14	
Oxyuris vermicularis in........	3	
Amœba coli in................	3	
Tænia saginata in............	2	
Tænia solium in..............	2	

Newton Evans (*Southern Medical Journal*, Nov., 1911) examined the stools of 122 children in public institutions of Tennessee and found worms in 60 children, or nearly 50%, though no symptoms were present. The order of frequency was as follows:

1. Hook worm (39 cases).
2. Round worm.
3. Whip worm.
4. Dwarf tape worm.
5. Pin worm.

Ascaris lumbricoides has usually a thick, wavy ("mammillated") shell; but this is not always seen, and in its absence the egg is dis-



Heterophyes heterophyes.

Distoma felineum.

Distoma sinense.

Fasciola hepatica.

Distoma buski.

Dictocœlium lanceolatum.

Tænia solium.

Tænia saginata.

Bilharzia hæmatobium.

Diplogonoporus grandis.

Bilharzia hæmatobium.

Dibothriocephalus latus.

Bilharzia hæmatobium.

Ascaris lumbricoides.

Oxyuris vermicularis.

Paragonimus westermani.

Tænia nana.

Ascaris lumbricoides.

Anchylostoma duodenale.

Uncinaria americana.

Strongylus subtilis.

Strongyloides stercoralis.

Trichuris trichiura.

DRAWINGS OF EGGS OF INTESTINAL PARASITES.
All are magnified 250. (After Looss.)

tinguishable from Uncinaria americana chiefly by the absence of the segmentation usually seen in the egg of the latter (see Plate II., *b*).

Trichuris trichiura (also called Triococephalus dispar) has a thick shell, very dark-stained, and apparently pointed and perforated at each end, instead of curving evenly over as the uncinaria egg does (see Plate II., *c*).

Bilharzia eggs are not at all uncommon in the fæces, though more often described in the urine, in connection with hæmaturia. In the urine the terminal spine at one end is their most characteristic feature (see Plate III). In the fæces the spine is usually at one side (see Plate III).

The other eggs are briefly described in the explanatory text accompanying Plate II.

II. The Spleen

Diseases of the spleen (abscess, malignant disease, cyst) are almost never recognized during life. It is for evidence of splenic enlargement as a factor in the diagnosis of diseases originating elsewhere that we investigate the splenic region as part of the routine of abdominal examinations.

Splenic enlargement is detected chiefly by palpation. Percussion plays a minor rôle in the determination of the organ's size, and should never be relied on in the absence of palpable evidence. Palpation is easy, provided the organ is enlarged sufficiently to project beyond the ribs without forced respiration, but much practice is needed when the enlargement is slight as in, for example, most cases of typhoid fever.

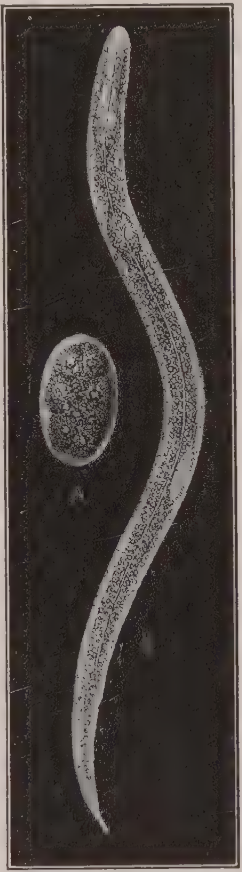


Fig. 214. — Strongyloides stercoralis. Magnified about 250 diameters. (After Thayer.)

1. *Palpation of the Spleen*

The co-operative action of both hands is as essential as in vaginal examination, and each hand must do the right thing at the right moment. The patient should be on his back, his head comfortably supported and his knees drawn up. The left hand, placed over the normal situation of the spleen, (*a*) draws the whole splenic region downward and inward toward the expectant finger-tips of the right hand, (*b*) at the same time the left hand should slide the skin and subcutaneous tissues over the ribs and toward the right hand (see Fig. 215), so as to leave a loose fold of skin along the margin of the ribs and give the palpating fingers a slack rather than a taut covering to feel through.

The right hand lies on the abdominal wall just below the margin of the ribs, and the fingers should point straight up the path down which the spleen is to move, *i.e.*, obliquely toward the left hypochondrium. With the hands in this position ask the patient to draw a full breath. Keep the hands still and do not expect to feel anything until near the end of inspiration. Then draw the hands slightly toward each other and dip in a little with the right finger-tips, so that if the spleen issues from beneath the ribs its edge will meet the finger-tips for an instant and spring over them as they rise from diving into the soft tissues (see Fig. 215).

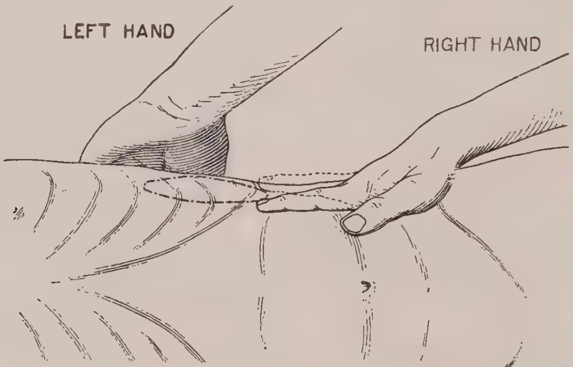


FIG. 215.—Position of the Hands in Palpation of the Spleen.

Some physicians have the patient lie on the right side, and, standing behind him, hook their fingers over the ribs in the left hypochondrium. In this way we may be able to feel the spleen at the end of a long inspiration, but I have seldom found this position as useful as that described above.

A hard, fibrous spleen (malaria) is much easier to feel than soft one (typhoid).

2. *Percussion of the Spleen*

Only when the edge of the spleen has been felt is it worth while to try to define its upper border by percussion. Normally there is dulness in the midaxillary line from the ninth to the eleventh ribs, corresponding to that part of the spleen that is most superficial. Its lower and posterior borders cannot be defined; its anterior edge is approximately in the midaxillary line (see Fig. 59). If this small area of dulness is enlarged upward and forward, and if the edge has been felt below the ribs, it is probable that the increased area of dulness corresponds to an enlargement of the organ.

3. *Causes of Splenic Enlargement*

Slight enlargement of the spleen can often be detected in:

1. Rickets and other debilitating conditions of childhood with or without anæmia.
2. Malaria.
3. Typhoid fever.

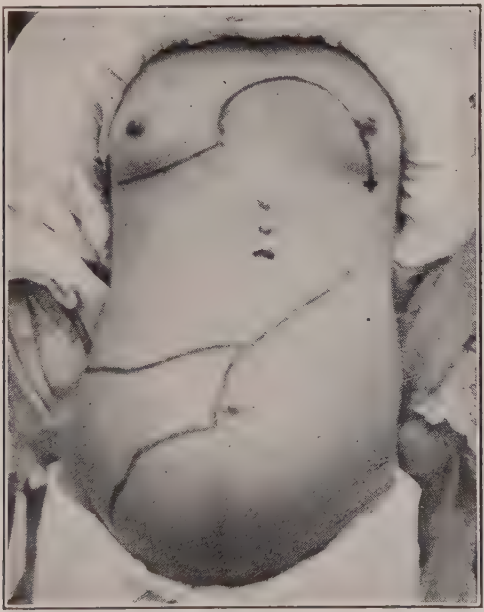


FIG. 216.—Splenic leucæmia.

In other acute infections slight enlargement can usually be made out post mortem, but not during life.

In a series of 100 cases of marked splenic enlargement studied in the Massachusetts General Hospital I found the following types:

1. Leucæmia—35 cases.
2. Hepatic cirrhosis—30 cases.
3. Malaria—8 cases.
4. Hodgkin's disease—6 cases.
5. "Splenic Anæmia"— 4 cases.
6. Syphilis—2 cases.
7. Polycythæmia—2 cases.
8. Amyloid—1 case.
9. Unknown Cause—13 cases.

Rarer causes are abscess, tuberculosis, malignant disease, pernicious anæmia, hydatid, and Leishman-Donovan disease.

Differences Between a Large Spleen and Tumors (of the kidney or other organs).—A large spleen is easily recognized after a little practice. *As it enlarges it keeps its shape* and advances obliquely across the belly toward the navel or (in marked cases) beyond it.

It is always *hard* and *smooth* of surface, although the edge nearest the epigastrium shows one or more *notches* which are very characteristic. The *edge is sharp*, never rounded, and the whole organ is very *superficial*, being covered only by the belly walls, so that if we inflate the *colon* (by forcing air into the rectum with a Davidson syringe), it *passes behind the spleen* and does not obliterate its dulness.

Tumors of the kidney fill out the flank, and an impulse can be transmitted to the lumbar region by bimanual palpation. They have no sharp edge or notches, are often irregular of surface, and not so superficial. The inflated colon passes in front of a tumor of the kidney and obliterates the dulness due to it.

All these differences hold for any other tumors likely to be confused with an enlarged spleen.

4. *Differential Diagnosis of the Various Causes of Splenic Enlargement*

In *children* splenic enlargement without fever or leukæmic blood changes is to be classed as a manifestation of general debility. It has no special connection with any type of anæmia, though anæmia is often seen with it.

In *typhoid* the fever with the Widal reaction and blood culture are generally sufficient to make clear the cause of the splenic enlargement; in active *malaria* the blood parasites are always demonstrable, and in chronic cases the history and the locality are significant.

Hepatic cirrhosis (and Banti's disease) should show evidences of portal stasis (ascites, jaundice, hæmatemesis).

Splenic anæmia means simply an anæmia of unknown origin associated with an enlarged spleen.

Leukæmic enlargement of the spleen is easily recognized by the characteristic blood picture.

Hodgkin's disease (lymphoma) shows enlarged glands in the neck, axillæ, and groins, with normal blood. Histological examination of an excised gland is necessary for diagnosis.

Amyloid can be suspected if there is a history of syphilis or chronic suppuration (hip abscess, etc.).

III. Diseases of the Kidney

1. *Incidence of Renal Disease* (*Massachusetts General Hospital*, 1870–1905)

Acute nephritis	200
Chronic glomerulo-nephritis	417
Chronic vascular nephritis	250[1]
Amyloid nephritis	9
Floating kidney	227
Stone in the kidney	145
Malignant disease	42
Tuberculous kidney	41
Renal sepsis (pyonephrosis, pyelitis and suppurative nephritis)	54
Perinephritic abscess	35
Hydronephrosis	19
Cystic kidneys	10
Total	1,449

We get evidence of diseases of the kidney in six ways:

1. By study of the constitutional symptoms,—fever, leucocytosis, anæmia, uræmia, dropsy, blood pressure and cardiac hypertrophy.
2. By cystoscopy, the ureteral catheter, and pyelography.
3. By examination of the urine.
4. By external examination and radioscopy of the region of the kidney.
5. By study of the non-protein nitrogen and other retention products in the blood.
6. By the "Red" test (Phenolsulphonephthalein).

Local examination acquaints us with the presence of tenderness and tumor.

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[1] Seven hundred and seventy-five other cases of "nephritis" not further specified.

(*a*) *Tenderness* is often present in renal abscess (tuberculous or non-tuberculous), pyelitis, and perinephritic abscess, less often in connection with nephrolithiasis, occasionally in hydronephrosis and malignant disease. A floating kidney may have an exquisite and peculiar sensitiveness to pressure, which differs from ordinary tenderness.

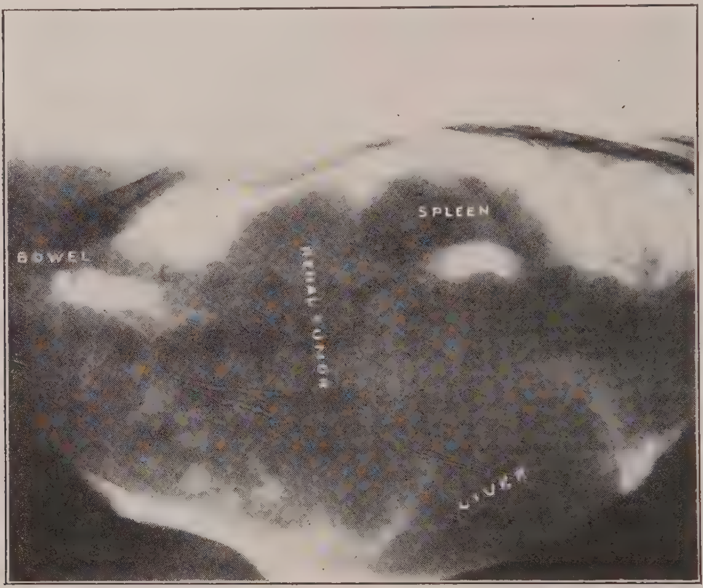

Fig. 217.—Renal tumor made visible by pneumoperitoneum. Patient lies on his right side.

(*b*) *Tumor* in the kidney region may occur in *abscess* in or around the kidney (including *tuberculosis of the kidney* and pyonephrosis), *malignant disease, hydronephrosis, and cystic kidney.* The latter members of this list afford examples of the largest tumors associated with the kidney.

2. *Characteristics Common to Most Tumors of the Kidney*

Renal tumors are best felt bimanually, one hand in the hypochondrium and the other in the region of the kidney behind, with the patient in the recumbent position. In this way the tumor may often be grasped and an impulse transmitted from hand to hand. It is usually round and smooth, often very hard, less often fluctuating. It descends slightly with inspiration. If the colon is inflated by forcing air into the rectum with a Davidson syringe, resonance appears in front of the tumor; this serves to distinguish it from tumors of the spleen which are pushed forward by the inflated colon as it passes behind them. Tumors of the kidney never present a thin and sharp edge, like that of the spleen. Occasionally they are irregular and nodulated—a condition almost never found in the spleen. It must be remembered that *an enlarged kidney may be the sound kidney* hypertrophied in compensation for disease on the other side.

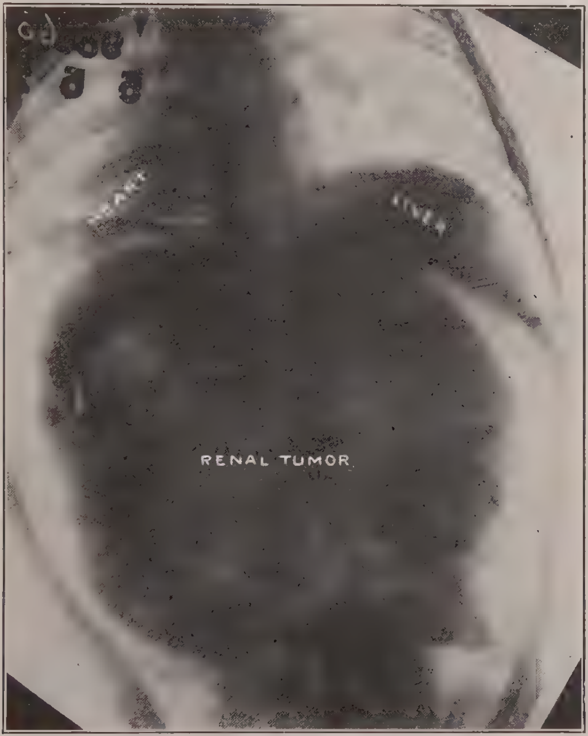

Fig. 218.—Pneumoperitoneum outlining renal tumor. Body seen from behind.

(*a*) *Malignant disease of the kidney*, usually a hypernephroma, makes up with cystic kidney the great bulk of the large abdominal tumors occurring in childhood, but is also not uncommon in adults.

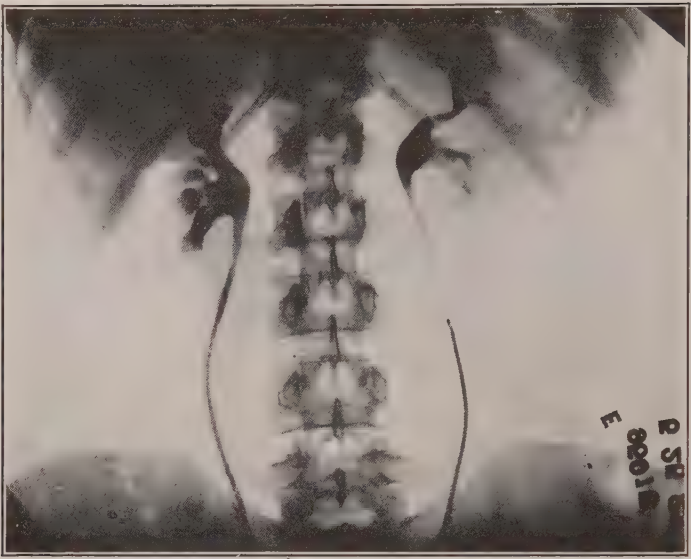

FIG 219.—Pyelograph of normal renal pelves.

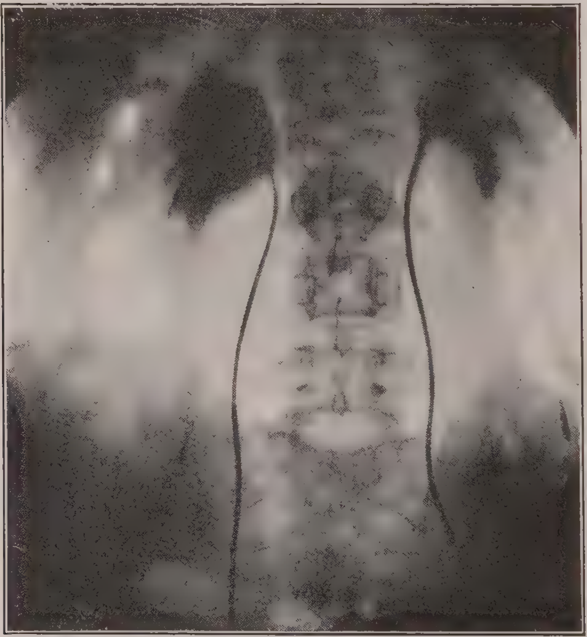

FIG. 220.—Bilateral hydronephrosis.

The characteristics of the tumor are those already described. There is usually pain, hæmaturia, emaciation, and anæmia sometimes leucocytosis, but small tumors at some distance from the renal pelvis are symptomless and unrecognizable. Pyelography may show distortion of the renal pelvis. *Metastases—especially bone metastases—are often the first evidence of the disease.*

(*b*) *Hydronephrosis and cystic kidney* may be indistinguishable from each other unless the hydronephrosis is intermittent and disappears with a great gush of urine, or unless the cystic kidney is bilateral—as indeed, is usually the case. In both diseases a smooth, round rumor forms in the loin and hypochondrium, usually without much constitutional disturbance and very frequently with a urine like that of contracted kidneys (see below) (see Fig. 222). Pain and tenderness are slight. In many cases the tumor is astonishingly hard and often gives no sign of fluctuation. With cystic kidney it may be coarsely lobulated. Like other tumors of the kidney it descends slightly on inspiration. Cystic kidneys are often congenital, but usually produce no symptoms until they have attained a considerable size, and hence are often overlooked or discovered accidentally.

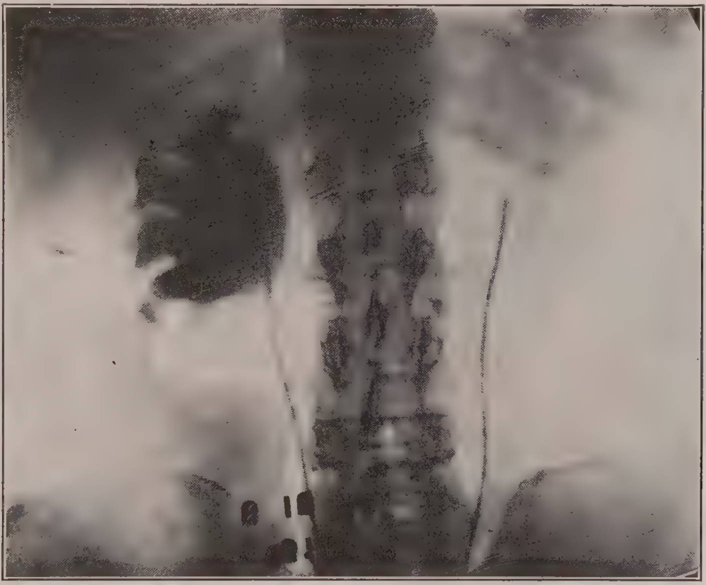

FIG. 221.—Pyelograph of hydronephrosis.

In hydronephrosis the diagnosis may be assisted by etiological hints, such as an abnormal degree of mobility of the kidney on the affected side, a history of renal colic with or without hæmaturia, or a prostatic obstruction. Comparatively slight degrees of dilatation or distortion of the renal pelvis and their relation to kinking of the ureter may be made out by the use of pyelography. This method is of great practical value in the diagnosis of hydronephrosis, pyonephrosis, pyelitis (which shows dilatation of the pelvis), renal tuberculosis and tumors, cystic kidney, hydroureter and ureteral obstruction, and for other purposes.

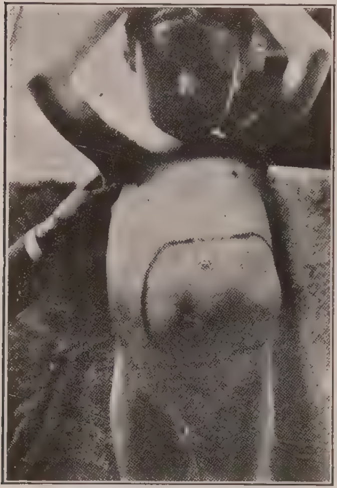

FIG. 222.—Left Hydronephrosis. A, Area of dull irregular resistance.

(*c*) *Perinephritic abscess* usually works its way to the surface in the back, between the crest of the ilium and the twelfth rib. This was the situation of the external tumor in 25 out of 35 cases recorded at the Massachusetts General Hospital. A tender swelling appears at the point just described, sometimes with redness and heat, and almost always with fever, chills, leucocytosis, and some emaciation. The urine may show nothing abnormal or may show the evidence of cystitis, of concomitant nephritis, or, rarely, of an abscess within the kidney itself. Perinephritic abscess often remains latent for weeks or months, and the amount of pus accumulated may be a quart or more.

(*d*) *Abscess of the kidney*, including tuberculous, suppurating kidneys and pyonephrosis, may produce a smooth rounded tumor in the hypochondrium and loin. It has the characteristics common to most renal tumors (see last page), but is usually distinguishable by:

1. The etiology (cystitis, stone in the kidney, tuberculosis, pyæmia). In acute cases, however, there is often no discoverable cause.

2. The presence of renal pyuria.

3. The presence of fever and leucocytosis. Persistent urinary frequency, especially nocturnal, in a young adult suggests renal tuberculosis. Animal inoculation with the urinary sediment obtained by ureteral catheter is essential to confirm diagnosis. *Bladder irritation is usually the first symptom of renal tuberculosis.*

(*e*) *Floating Kidney; Displaced and Movable Kidney.*—The tip of the right kidney is palpable in most thin persons with loose belly walls. If the whole organ is palpable but not movable, we speak of it as *displaced.* If the range of mobility is relatively great we call it *floating;* if relatively slight we call it *movable.* With bimanual palpation (as described above) we exert pressure just at the end of a deep inspiration and maintain it. During expiration something smooth and round may then be felt to slip upward between our hands toward the ribs. If the kidney "hides" behind the ribs, have the patient sit up, cough, and breathe deeply; then repeat the bimanual palpation as he lies on his back. Very movable or floating kidneys may be found far from their normal home, and are then recognized by: 1. Their size, shape, and slippery feel. 2. The sickening pain produced by pressure. 3. The possibility of replacing them.

3. *Renal Colic and Other Renal Pain*

Typical *renal colic* is *paroxysmal,* like all colics; that is, an attack begins suddenly, ends suddenly, and lasts but a few hours or less. The pain usually begins in the back, over the kidney, and follows the course of the ureter to the groin. During an attack the testicle on the affected side may be tender and drawn up tightly by contraction of the cremaster.

When associated with hæmaturia or pyuria, with or without sudden stoppage of water during an attack and without any general or constitutional symptoms between attacks, renal colic is suggestive of stone in the pelvis of the kidney; but similar attacks may occur with other surgical diseases of the kidney (tuberculosis, neoplasm, kinking of the ureter), etc., when blood clot or pus gets wedged into the ureter.

From *biliary colic* it may be distinguished by the (*a*) different situation of the pain, (*b*) by the presence of blood or pus in the urine, and (*c*) the absence of jaundice in this or a former attack.

In *intestinal colic* the pain shifts its position frequently and is associated with noises produced by wind in the bowels, or with diarrhœa or constipation.

Renal pain, not colic, may occur in almost any disease of the kidney *except nephritis*, and is characterized by its situation over the kidney and by the lack of any connection with muscular movements (lumbago), with spinal movements (hypertrophic arthritis), or with the sacro-iliac joint.

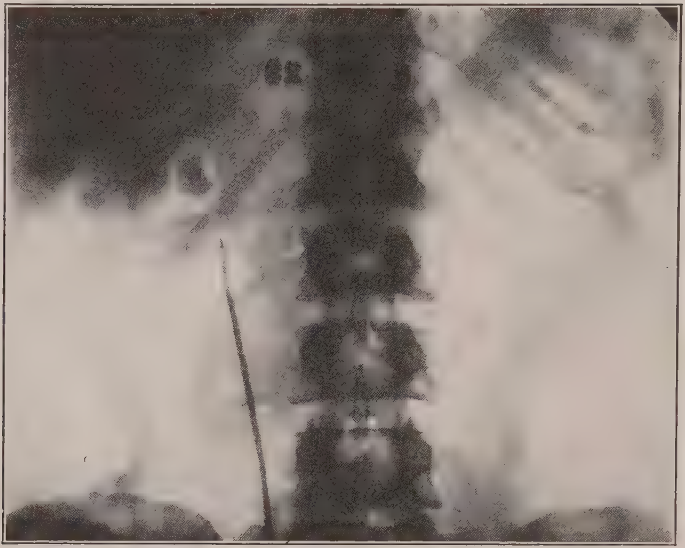

FIG. 223.—Stone in the kidney.

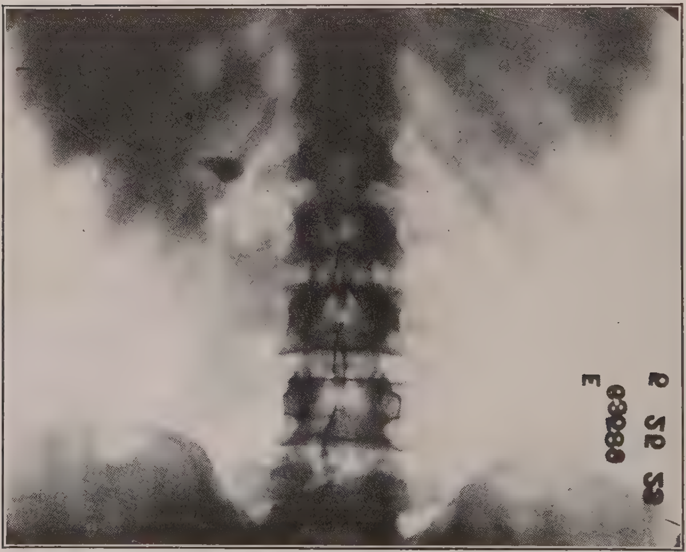

FIG. 224.—Stone in the kidney.

I have now described what seems to me most important in the *local external* examination for kidney disease, and have mentioned, along with the different lesions producing tumor, the general constitutional manifestations which are of assistance in diagnosis. Aside from the *local* and the *constitutional* evidence of renal disease (high blood pressure and enlarged heart), we have only the evidence afforded by the urine, to which I now pass on.

4. *Examination of the Urine*

The urine as passed *per urethram* is a resultant and reflects the influence of many different organs and surfaces. Thus disturbances of metabolism, such as diabetes, intoxications (lead, arsenic), disease of the heart, liver, and intestine, febrile conditions, infective or malignant disease of any part of the urinary tract (kidney, ureter, bladder, or urethra), as well as the different types of nephritis, all affect the urine, though hardly any of them produce pathognomonic changes in it. In this section I shall consider the urine as a piece of evidence in the diagnosis of kidney disease, and only in contrast with this will its characteristics in extrarenal troubles be mentioned briefly.

The most essential features of the urine in the diagnosis of kidney disease are:

1. The amount passed in twenty-four hours, measuring separately the portions passed at night (8 P.M. to 8 A.M.) and in the daytime (8 A.M. to 8 P.M.).
2. The specific gravity.
3. The looks (optical properties).
4. The "red test" (see page 418).
5. The reaction to litmus.
6. The presence of blood, pus, or tubercle bacilli.
7. The presence or absence of albumin and sugar.

Much less important than these is the presence or absence of casts, cells, crystals, etc.

(a) *The Amount and Weight of the Urine*

The twenty-four-hour amount concerns us chiefly in diabetes, the different types of nephritis, and in cardiac decompensation.

Polyuria occurs in health after the ingestion of large quantities of water, and sometimes in conditions of *nervous strain*. In disease it characterizes both forms of *diabetes*, *contracted kidneys* (primary, secondary, or arterio-sclerotic), and is seen during the *convalescence from acute nephritis and from various infectious diseases.* It also occurs in the early stages of renal tuberculosis or when continuous drainage (catheter) is established in cases of prostatic obstruction. In diabetes of either form several quarts or even gallons may be passed. In contracted kidneys the increase of urine occurs very largely at night, so that the amount may be double that passed in the day-time, just reversing the conditions of health.

Oliguria or scanty urine occurs in health when the amount of water ingested is small or when water is passed out of the body abundantly through the skin or by the bowels (diarrhœa). In disease oliguria or absolute suppression of urine (*anuria*) occurs at the beginning of *acute nephritis* and as a result of occlusion of one or both ureters by *stone or malignant disease.*[1] Remarkable examples of anuria also occur in *hysteria. Infectious fevers and cachectic states* often diminish the secretion of the urine by one-half or more.

The *specific gravity* is usually low with polyuria and high with oliguria, but in diabetes mellitus the presence of the sugar gives us polyuria with high specific gravity.

Total Urinary Solids.—By multiplying the last two figures of the specific gravity by the number of ounces of urine passed in twenty-four hours and the product by 1.1, we get a figure representing the total urinary solids in grains, with accuracy sufficient for clinical diagnosis. Thus if 30 ounces of urine are passed in 24 hours and the gravity is 1.020, then 20 × 30 × 1.1 = 660 grains.

(*b*) *Optical Properties*

Color.—Dilute urines (polyuria) are generally pale, and concentrated urines (oliguria) high in color. A dark or brownish tint in the urine is generally produced by bile, by blood pigment, or as a result of certain drugs—carbolic acid, coal-tar preparations, and salol. If the color is due to bile, a bright canary yellow appears in the foam after shaking up a little of the urine in a test tube. No other tests for bile are necessary. Urines darkened by blood pigment show abundant blood corpuscles in the sediment;[2] when the color is due to drugs we can usually learn this fact from the history.

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[1] It is a remarkable but well-attested fact that when one ureter is suddenly blocked both kidneys may stop secreting for the time. Yet when one kidney is gradually destroyed as in tuberculosis, the other hypertrophies so as to assume the function of both.

[2] Except in some cases of hæmoglobinuria.

Turbidity in alkaline urine is usually due to the presence of bacteria. In acid urine it is produced in a great majority of cases by amorphous urates, and disappears on heating the urine, while the turbidity due to bacteria is unaffected by heat. Normal urine may be turbid and alkaline, owing to the presence of insoluble carbonates and phosphates, but clears on the addition of acetic acid. Hence turbidity, not removed by heat or acetic acid, is almost always due to bacteria and pus, *i.e*, to cystitis, pyelonephritis, or both.

Shreds seen floating in the urine and found to be composed mostly of pus are presumptive evidence of urethritis, and practically always of gonorrhœa.

The *gross sediment* as seen by the naked eye amounts in health to nothing more than a slight cloud, which settles in the lower part of the vessel containing the urine. This cloud is somewhat denser in women than in men, owing to the presence of vaginal detritus. When the gross sediment amounts to anything more than this, it is almost invariably made up of (*a*) *pus*, (*b*) *blood*, or (*c*) *urates*. The latter are dissolved on heating. Pus has usually its ordinary yellow color and general appearance. Blood may be somewhat lighter or somewhat darker than under ordinary conditions, but is usually recognized without difficulty.

(*a*) *Significance of these Sediments*

A *urate sediment* means nothing more than a concentrated urine standing in a cold room. In the winter-time patients often bring us, in great alarm, a bottle of milky or fawn-colored and turbid urine, which is not in any way abnormal. The urates have been precipitated over night by the low temperature of the bedroom.

Pyuria, or gross pus in the urine, is oftenest seen in cystitis and less often in pyelonephritis and renal suppurations, tuberculous or pyogenic. The pus occurring in gonorrheal urethritis is usually much less in quantity than that coming from the bladder or kidney, and can be distinguished by the local signs of gonorrhœa. Leucorrhœal pus can be excluded by withdrawing the urine by catheter. The rupture into the urinary passages of an abscess from the prostate or any part of the pelvis may produce a profuse but transient pyuria.

After excluding gonorrhœa, leucorrhœa, and abscess, which can usually be done with the help of a good history and a catheter, we have left *cystitis* and *renal suppurations*, which it is very important and sometimes difficult to differentiate. In both we have the frequent and painful passage of small quantities of a urine which is in no way remarkable except in containing large amounts of pus and bacteria. Cystoscopy is often essential. In the vast majority of cases "cystitis" is secondary to some other disease above or below the bladder—*e.g.*, to prostatic obstruction, renal tuberculosis, etc.

In many cases the differentiation may be accomplished as follows: Have the patient save for twenty-four hours the urine voided at each passage *in a separate bottle* (all of the bottles being of uniform size), and mark each bottle with the hour at which it was filled. Then arrange the specimens in a row, beginning with that passed earliest and ending with that passed last. Now if the case is one of cystitis without involvement of the kidney, the amount of pus that settles is practically the same in each bottle (allowing for differences in the amount of urine in the different bottles). But if the pus comes from the kidney, it is almost always discharged intermittently, and hence some of the bottles will be almost free from sediment, while in a group of the others the amount of pus increases as we pass along the line, reaches a maximum in one or two bottles, and decreases again in those representing the later acts of micturition.

Pus from the bladder is generally alkaline, although in tuberculosis it may be acid; pus from the kidney is generally acid. When both organs are involved, as is frequently the case, we have a mixture of the characteristics of both types of pyuria, and cystoscopic examinaoion with or without catheterization of the ureters is usually necessary.

5. *Pyuria*

In *renal pyuria* we often have local signs in the renal region (tumor and tenderness), a history of renal colic, and decided constitutional symptoms.

In *vesical pyuria* we have vesical pain, often tenesmus, no renal pain or tumor, and usually slighter constitutional symptoms. The amount of squamous epithelium (see below) is sometimes larger in cystitis than in renal suppurations, but no reliable inferences can be drawn from the size or shape of the cells.

To determine whether pus from the bladder or the kidney is tuberculous or non-tuberculous in origin, we usually inject the sediment into a guinea-pig, which develops tuberculosis or not according to the nature of the pus injected. This method is much more reliable than the bacteriological examination of the sediment, for besides the tubercle bacillus other bacilli which retain fuchsin and resist decolorization by strong acid and by alcohol occasionally occur in the urine.

6. *Hæmaturia*

In searching for the source of the blood we must be sure to exclude the female genital organs. Menstrual blood and uterine bleeding from various other causes often contaminate the urine, and must be excluded by using a catheter.

The causes of true hæmaturia, arranged approximately in the order of frequency, are:

1. Prostatic congestion or irritation.
2. Stone in the kidney (less often vesical stone).
3. Tumors of the kidney or bladder.
4. Acute nephritis and acute hemorrhage in chronic nephritis.
5. Tuberculosis of the kidney or bladder.

Less common causes are: floating kidney, hydronephrosis and cystic kidneys, animal parasites in the urinary passages, poisons (turpentine, carbolic acid, cantharides), hemorrhagic diseases (purpura, scurvy, leukæmia), trauma and renal infarction. *In nearly one-fourth of all cases no cause can be found.*

In *cystitis* there are bladder symptoms—pain, tenesmus, frequent and painful micturition. The blood is mixed with pus and epithelium, and is especially abundant in the urine passed near the end of the act of micturition. If the bladder is irrigated it is hard to get the wash-water clear. Cystoscopy demonstrates or upsets the diagnosis and also serves to show some other disease to which the cystitis is secondary. Distrust all diagnoses of "primary cystitis."

In *renal stone* there are no bladder symptoms to speak of, the blood is pure and thoroughly mixed with the urine, and if the bladder is washed out the final wash-water is clear. There is often renal colic (see p. 411) and sometimes the passage of stones or gravel by urethra. X-ray evidence is usually conclusive.

In *acute nephritis* the blood is rarely fresh, generally dark chocolate in color. The twenty-four-hour amount of urine is small, and albumin and casts (see below) are abundant. General œdema is common. Local symptoms in the kidney or bladder are absent. Most cases of "acute nephritis" in adults turn out, on careful study, to represent acute exacerbations of chronic nephritis.

In *renal tumor and especially in renal tuberculosis* we have often pyuria and the local and constitutional evidences above described (page 410), with marked and early bladder symptoms (*even when the bladder is not diseased*).

Tumors of the *bladder* need cystoscopy for diagnosis.

In the diagnosis of the rarer forms of hæmaturia we rely chiefly on the history (trauma, poisons ingested) and on the evidences afforded by cystoscopy and general physical examination.

7. *The "Red Test" (Phenolsulphonephthalein) for Renal Function*

Of the many "functional" kidney tests introduced in recent years that of Rowntree and Geraghty is by far the most useful for clinical purposes. One cubic centimeter of the standard *sterilized* solution of phenolsulphonephthalein[1] is injected subcutaneously into the back or thigh. Two hours later the patient passes urine which is then made alkaline with sodic hydrate and diluted up to 1 liter. The degree of red color in the resulting fluid is then compared in a test tube with a scale of standard test tubes representing 90 per cent., 80 per cent., 70 per cent., etc., of the amount of coloring matter originally injected. Such a scale of test tubes can be cheaply made by any one who has access to a Dubosc colorimeter. The scale can also be bought for a few dollars from Joseph Godsoe, Massachusetts General Hospital, Boston. If there is prostatic obstruction or urethral stricture the patient should be catheterized when the "red" is injected and at the end of two hours.

Normal young adults excrete 65 per cent. to 90 per cent. of the color injected within 2 hours. In nephritis and decompensated heart disease the color output may fall to 5 per cent. or to zero. In elderly people confined to bed 20 to 40 per cent. may be considered normal and does not indicate nephritis. Allowing for age and activity the test is a valuable measure of the kidney's general excretory function.

8. *Chemical Examination of the Urine*

(*a*) *The Reaction of the Urine*

The reaction of normal urine is acid to litmus, except temporarily after large meals. Its acidity becomes excessive in fevers or occasionally without any known cause.

Alkaline urine has generally an ammoniacal odor and suggests cystitis. As a result of decomposition and bacterial fermentation all urine becomes alkaline (ammoniacal) on standing exposed to air.[1] Occasionally we find urine alkaline from fixed alkali and without known cause.

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[1] Any dealer in medical supplies can furnish it.

The *value* of the litmus test is chiefly as *prima-facie* evidence of stasis in the bladder and cystitis. Occasionally tuberculous cystitis and the first stages of any variety of cystitis are associated with acid urine, but in most cases lasting over a week ammoniacal fermentation and alkalinity appear.

(*b*) *Albuminuria and the Tests for It*

Serum albumin is the only variety of clinical importance, and for this but two tests are necessary: (1) Nitric-acid test; (2) test by boiling.

The *nitric-acid test* is best performed in a small wineglass. After filling this half full of urine, insert a small glass funnel to the bottom of the urine and gently pour in concentrated nitric acid. If albumin is present, a white ring forms at the junction of the acid with the urine, either immediately or in the course of ten minutes. If carefully performed this test is delicate enough for all clinical purposes, but since some of the albumoses give a similar precipitate, the boiling test should be used as a control whenever a positive reaction is obtained with nitric acid. None of the other rings, observable above or below but not at the junction of the acid with the urine, is of any clinical importance.

The Boiling Test.—To half a test tube full of urine add three or four drops of dilute acetic acid, and boil the upper three-quarter inch of the urine. If albumin is present a white cloud appears. If the Bence-Jones body is present, a white cloud appears on heating, *disappears on boiling, and reappears on cooling*. In performing this test the addition of acetic acid as above described is absolutely necessary to prevent error.

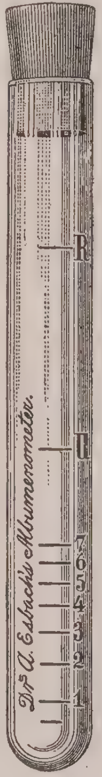

FIG. 225.—Esbach's Albumenometer.

For the detection of albumin no other tests are needed. For its approximate quantitative estimation, Esbach's method is the best.

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[1] Simultaneously a dark-brown color rarely appears: *alkaptonuria*, a fact at present of no clinical significance except that such urines reduce Fehling's solution and may be mistakenly supposed to contain sugar.

Esbach's Method.—A special tube (see Fig. 225) is employed. Urine is poured in up to the mark "U," and then Esbach's reagent[1] up to the mark "R.' The tube is then corked, inverted about half a dozen times, and set aside for twenty-four hours. A precipitate falls and the amount per mille is then read off on the scale etched upon the tube. If the urine is not acid it must be made so with dilute acetic acid, and unless its specific gravity is already very low it should be diluted once or twice with water so as to bring the gravity below 1.008. After such a dilution we must, of course, multiply the result obtained by a figure corresponding to the dilution. The method is not accurate, but is probably accurate enough for practical purposes, if all tests are made at approximately the same temperature.

(*c*) *Significance of Albuminuria*

It is important to realize that albuminuria very often occurs without nephritis and that nephritis occasionally occurs without albuminuria. Among the more important types not due to kidney disease are the following: (1) Juvenile albuminuria; (2) febrile albuminuria; (3) albuminuria from renal stasis; (4) albuminuria due to pus, blood, bile, or sugar in the urine; (5) toxic albuminuria.

Besides these, there are a good many cases of albuminuria occurring in diseases of the blood, after violent exertion, after epileptic attacks, and without known cause. Many of the juvenile group occur only when the patient is on his feet and are absent as long as the patient lies down (*orthostatic albuminuria*); others occur irregularly or at regular intervals (*cyclic albuminuria*). Most of these cases appear at adolescence and all pass off without nephritis ever developing. Nearly one in ten college freshmen shows this harmless albuminuria.

Exclude the juvenile cases, fever, circulatory disturbance, anæmia, poisons—such as cantharides, turpentine, carbolic acid—and deposits of blood or pus in the urine, before deciding that any case of albuminuria is due to nephritis. In general, it is a good rule not to attribute albuminuria to nephritis unless there is other and more convincing evidence in the physical characteristics of the urine and in the other organs of the patient. If the 2-hour amount and the gravity are approximately normal, and if there is no œdema, no increased blood pressure, no cardiac hypertrophy, no uræmic manifestations, and nothing alarming in the sediment of the urine, we should not diagnose nephritis. I shall discuss this point further in the section on the examination of the sediment (see page 423). It will be noted that *practically all the types of albuminuria not due to nephritis are transient*, while, with the exception of certain stages of chronic nephritis, the albuminuria of nephritis is as permanent as the nephritis itself. On the other hand, many cases of nephritis (so proved by autopsy) show no albuminuria for long periods. They are then to be recognized by the low fixed gravity, the evidence of high blood-pressure and by its results.

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[1] Esbach's reagent: Picric acid, 10 gm.; citric acid, 20 gm.; distilled water, 1,000 c.c.

(*d*) *Significance of Albumosuria*

The *Bence-Jones body* is very constantly present in the urine of cases of multiple myeloma. It has no known importance in any other disease. Deuteroalbumoses have no clinical significance.

(*e*) *Glucosuria and Its Significance*

For glucose in the urine we need but one qualitative and one quantitative test, viz., Fehling's test and the fermentation test.

1. *Fehling's Test.*—Mix in a test tube equal parts of a standard solution of copper sulphate[1] and a standard solution of alkaline tartrates,[2] and add to this mixture an equal amount of urine. Mix and heat nearly to boiling. The amount of error entailed by boiling is slight and unimportant, but the only advantage of boiling is a slight saving of time. If sugar is present a yellow or reddish-yellow precipitate occurs, either at once or (if the amount of sugar is very small) after the urine has cooled. Fehling's solution may also be used for quantitative estimation of sugar, but it is more convenient to use:

2. *The Fermentation Test.*—Take the specific gravity of the urine as carefully as possible, and acidify it if necessary with acetic acid. Pour six or eight ounces of urine into a wide-mouthed vessel and crumb into it half a cake of fresh Fleischmann's yeast. Set the flask aside in a warm place, and after twenty-four hours test the supernatant fluid with Fehling's solution as above; if sugar is still present fermentation must be allowed to go on twenty-four hours longer. As

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[1] Made by dissolving 34.64 gm. pure $CuSO_4$ in water and then adding enough water to make 500 c.c.

[2] Made by dissolving 173 gm. Rochelle salts and 60 gm. sodic hydrate each in 200 c.c. of water, mixing the two solutions, and adding water to make 500 c.c.

soon as a negative reaction to Fehling's has been secured (whether in twenty-four or forty-eight hours), the specific gravity of the filtered urine is again taken.[1] It will be found lower than before the fermentation, and for every degree of specific gravity lost we may reckon that 0.23 per cent of sugar has been fermented out of the urine. Thus if the reading was 1.040 before fermentation and 1.020 afterward, we multiply the difference between these readings, 20, by 0.23, giving 4.6 per cent—the percentage of sugar.

Fehling's test should be applied to every urine examined; it takes but a minute or two. *When it shows a yellow or red precipitate, the fermentation test should also be tried;* and *if both tests are positive we shall run but a negligible risk in saying that glucose is present.* From the result of the fermentation test and the twenty-four-hour amount of urine, we can estimate the daily output of sugar through the urine.

Permanent glucosuria means diabetes mellitus. Transitory glucosuria may be due to a great many causes, among which are: (1) Diseases of the liver; (2) diseases of the brain, organic or functional, especially the latter; (3) infectious fevers; (4) poisons, especially narcotics (alcohol, chloral, morphine); (5) pregnancy; (6) exophthalmic goitre.

Experimental ("alimentary") glucosuria or levulosuria can be produced in many of these same diseases by giving the patient 100 gm. of glucose or levulose in solution.

The differential diagnosis of the cause of glucosuria depends on the recognition of one of the above conditions. The other sugars occasionally found in the urine (levulose, lactose, pentose, etc.), are of no clinical importance.

(*f*) *The Acetone Bodies*

Acetone, Diacetic and Beta-Oxybutyric Acids

1. *Test for Acetone.*—To about one-sixth of a test tube of urine add a crystal of sodium nitroprusside, and then NaOH to strong alkalinity. Shake and add to the foam a few drops of glacial acetic acid. *A purple color shows acetone.*

2. *Test for Diacetic Acid.—A Burgundy red color* when a strong aqueous solution of ferric chloride is added to fresh urine (*not* previously boiled) in a test tube. If this reaction is well marked beta-

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[1] The room temperature must be approximately the same as at the time of the previous reading.

oxybutyric acid is probably also present, but we possess no clinical test for the latter substance.

Significance of the Acetone Bodies.—Diminished utilization of carbohydrate food by the body is usually the cause of the appearance of these bodies in the urine. This may occur: (*a*) Because sufficient carbohydrates are not *eaten* (starvation, rectal alimentation, fevers, etc.). (*b*) Because they are not *absorbed* (vomiting, diarrhœa, etc.). (*c*) Because they are not *assimilated* (diabetes), and rarely for other reasons.

(*g*) *Other Chemical Tests*

The information to be derived from testing for indican, for the amounts of urea, uric acid, chlorides, phosphates, and sulphates, does not seem to me sufficient to justify the time spent. The same is true of the diazo reaction. The guaiac test for blood, described above (see p. 371) in connection with the examination of gastric contents and fevers, is also of value in the urinary examination.

Simon's lucid arguments for the value of the *indican* test have not been borne out by my experience with it in diagnostic puzzles. The tests for *urea* and *uric acid* are of value only when we possess a knowledge of all the factors governing their excretion, knowledge which in clinical work we almost never have. Diminution or absence of the urinary chlorides in pneumonia is not constant, and occurs in many other infections (typhoid, scarlet fever, etc.). The diazo reaction is nearly constant in typhoid, but is occasionally found in so many other febrile and cachectic states that most clinicians have ceased to rely on it. Its value in the prognosis of phthisis is slight. I believe that the general abandonment of the tests for the sulphates and phosphates have now led to the abandonment of the tests for urea, uric acid, indican, and the chlorides. The use of these tests gives the *appearance* of accuracy and scientific method in diagnosis—the appearance, *but not the reality*.

9. *Microscopic Examination of Urinary Sediments*

Methods.—A centrifuge is convenient, but not necessary. The sediment should be allowed to settle in a conical glass (see Fig. 226), whence a drop of it can be transferred to a slide by means of a pointed glass pipette. Close the upper end of this with the forefinger and introduce the pointed end into the densest portion of the sediment; next very slightly relax the pressure of the forefinger until urine and sediment flow into the lower one-half or three-fourths inch of the pipette. Then resume firm pressure with the forefinger, withdraw the pipette, wipe the outside of it dry, put its point upon a microscopic slide, and again *slightly* relax the pressure of the forefinger so as to let a small drop of urine and sediment run out upon the slide. Cover this drop with a seven-eighths inch cover glass, and examine it with a Leitz objective No. 5 or Zeiss DD.

The arrangement of the light is most important. The iris diaphragm should be closed until one can just distinguish the outlines of the cells and other objects in the field. If more light is admitted the pure hyaline casts will be invisible.

FIG. 226.—Conical Glass for Urinary Sediments.

Results.—The objects most often sought for in the sediment are: (*a*) Casts; (*b*) cells; (*c*) crystals; (*d*) animal parasites or their eggs.

1. *Casts.*[1]—Casts, or moulds of the renal tubules, may be homogeneous and transparent (hyaline, Fig. 227, 1) or may have attached to this matrix a variety of *granules*, *cells*, *crystals*, or *fat drops*. According to the variety of passengers carried down from the kidney on the casts, we call them *granular*, *brown-granular*, *cellular*, *blood*, *fatty*, or *crystal-bearing* casts (see Fig. 227, 2 and 3, and Fig. 228, 1, 2, 3 and 4).

Dense or *highly refractile casts*, colorless or straw colored, are occasionally seen, and are often given a variety of names quite unjustified by any knowledge of their composition (*e.g.*, "waxy,"[2] "fibrinous," etc.).

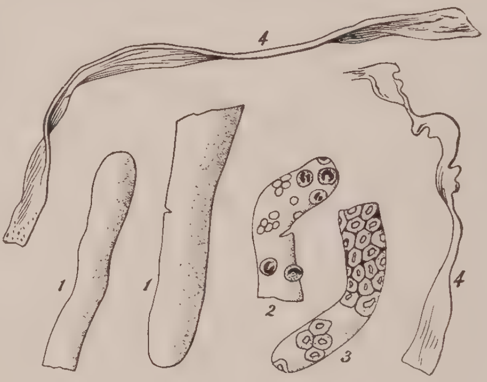

FIG. 227.—Casts. 1, Hyaline casts; 2 and 3, hyaline casts with cells and blood adherent; 4, "cylindroids."

From strands of mucus, foreign bodies, and other sources of error, true casts may be distinguished by the following traits:

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[1] Though I have here described casts first I believe that the finding of blood or pus in the sediment is of far more frequent and more considerable importance.

[2] Some dense, refractile casts give the amyloid reaction, but this does not indicate amyloid kidneys and has no known clinical significance.

(*a*) Their sides are parallel.

(*b*) One end is rounded; sometimes both ends.

Red corpuscles and other cells upon casts are to be recognized—the former by the size, shape, and, if fresh, by their color (pale straw); the latter by the presence of a nucleus.

Fat drops are spherical and very highly refractile, so that they seem to have a black line at their periphery.

Crystals can be recognized by their angles. They are very rarely of importance. When showers of oxalate crystals in large masses are associated with attacks of hæmaturia not otherwise explained, the crystals may be of some etiological significance.

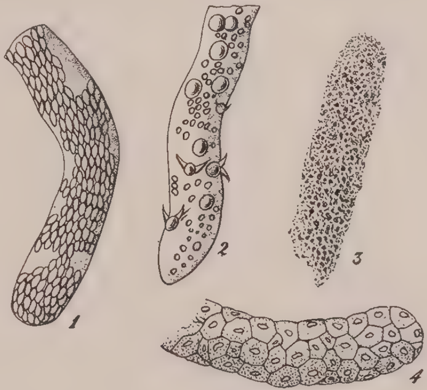

FIG. 228.—Casts. 1, Blood-casts; 2, fatty casts; 3, granular casts; 4, cellular casts.

Other bodies on casts are called *granules*.

SIGNIFICANCE OF CASTS.—Casts may occur in health (unless we choose to class muscular fatigue as disease) as well as under any of the conditions giving rise to albuminuria (see page 420). They are usually more numerous in acute nephritis and in the acute exacerbations of chronic nephritis than in most other conditions. Any type of cast may occur in any type of nephritis, but

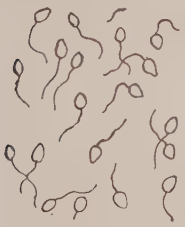

FIG. 229. Spermatozoa.

Cellular,[1] *blood*, and *brown-granular* casts are most often found in *acute nephritis*.

Fatty, *highly refracting*, or *dense* casts most often predominate in *chronic glomerular nephritis* ("diffuse" or "parenchymatous" nephritis).

Hyaline and *granular* casts may occur in any type of nephritis and in many other conditions (fatigue, renal stasis, etc.). In the urine of persons over fifty years of age the presence of a few hyaline and granular casts has no known clinical significance, and may probably be considered physiological.

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[1] "Cellular" is a better term than "epithelial," since we have no marks for recognizing renal epithelium or for distinguishing a renal cell from a lymphocyte.

Periods occur in the course of many cases of chronic contracted kidney when no casts can be found. If any occur they are usually of the hyaline and fine granular types.

2. *Free Cells in Urinary Sediment. A. Recognition.*—The presence of macroscopic pus or blood already alluded to may be verified by the microscope.

(*a*) Fresh red cells, lately freed from the blood-vessels, preserve their straw-yellow color. Their presence points to the recent effusion of blood, probably from the bladder, urethra, or renal pelvis.

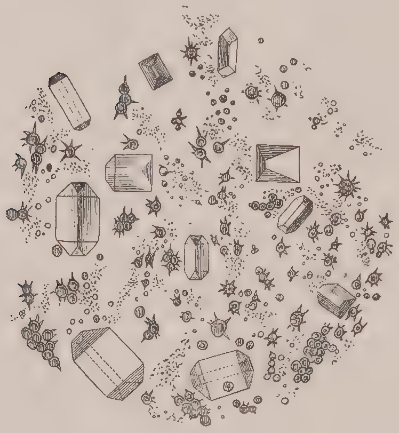

FIG. 230.—Crystals of Triple Phosphate (prisms) and Ammonium Urate (small spheres with spines.)

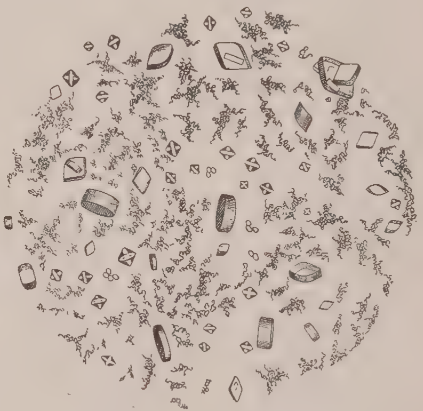

FIG. 231.—Crystals of Uric Acid (whetstone-shaped) with Calcic Oxalate (small octahedral) and Amorphous Urates.

(*b*) Abnormal blood, decolorized and shadowy red discs, can be recognized with practice by their size and shape. We may infer that they have remained some time in the urine and have probably come from the kidney.

(*c*) Pus is easily recognized as a rule by the presence of the familiar polymorphous nucleus in most of the cells. Should doubt arise, a drop of dilute acetic acid allowed to run under the cover glass will sharpen the outlines of the nuclei and facilitate their recognition.

(*d*) Spermatozoa (see Fig. 229) are often seen in the urine after coitus or nocturnal emissions. They are of no importance, except that when appearing in the urine of females they may afford valuable medico-legal evidence. They are easily recognized by their size and shape.

(*e*) Other varieties of cells need not be differentiated, since almost any of the varieties usually described (*squamous*, *spindle-shaped*, *caudate*, etc.) may come from any part of the urinary tract. *Renal cells* are not recognizable by our present methods of examination.

Any of the urinary cells may contain fat drops, but these have no special diagnostic significance.

B. Interpretation.—The significance of large quantities of blood or of pus in the urine has already been discussed (page 417). When recognizable *only* by the microscope they have no diagnostic value.

The presence of large numbers of cells not coming from the blood-vessels (squamous, spindle-shaped, etc.) is usually associated with cystitis, provided the accidental admixture of vaginal detritus is excluded. Pyelitis and renal suppurations may fill the sediment with similar cells, and only by other methods of examination (cystoscopy, ureteral catheterization) and by taking account of all the facts in the case can the differentiation be made.

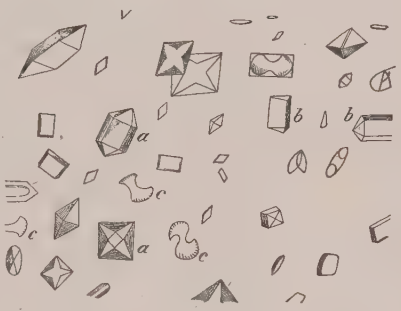

FIG. 232.—Calcic Oxalate Crystals.

3. *Crystals in Urinary Sediments* (see Figs. 230, 231 and 232).

The varieties oftenest seen are: (*a*) Triple phosphate (ammoniacal urine, cystitis); (*b*) ammonium urate; (*c*) uric acid; (*d*) calcic oxalate.

All of these varieties are colorless except the uric-acid crystals, which are usually light or dark *yellow* or yellowish-brown.

None of these have much significance in diagnosis. The first two merely confirm the evidence of urinary decomposition (usually from cystitis), afforded by the reaction, turbidity, and odor of the urine.

Uric-acid crystals, if present in great numbers in the urine when passed, suggest the search for macroscopic masses (gravel) and for other evidence of renal stone, but as a rule they are of no importance.

The same may be said of calcium oxalate. Oxaluria is one of the most persistent bugbears of the medical profession, but it is utterly harmless except in the rare cases in which it accompanies hæmaturia (see above) or gravel.

4. *Animal parasites or their eggs* are occasionally found in the urine, with or without hæmaturia and evidence of cystitis (see Figs. 233 and 234).

10. *Summary of the Urinary Pictures Most Useful in Diagnosis*

Aside from *polyuria, oliguria, hæmaturia,* and *pyuria,* which have already been discussed, the most important conditions in which the urine gives valuable diagnostic evidence are:

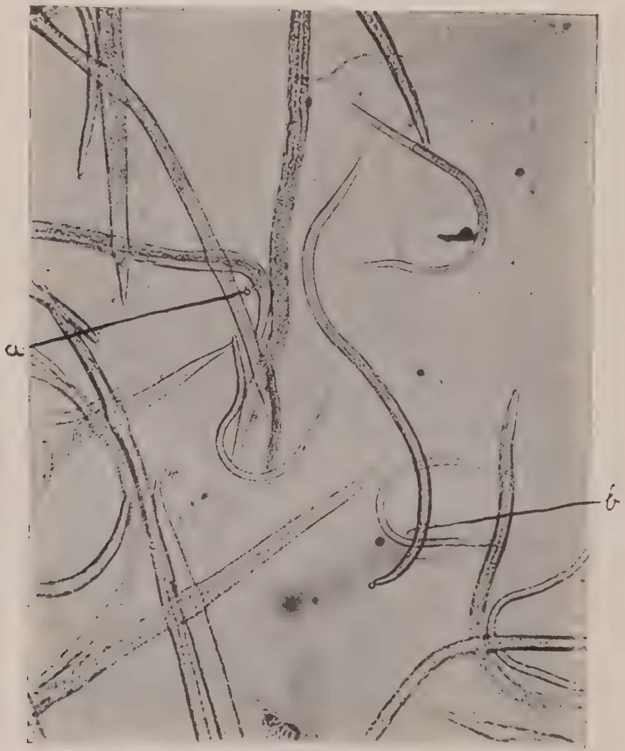

Fig. 233.—Vinegar Eels in Urine. (Billings.) *a*, Protruded hooks of male; *b*, top-shaped œsophageal enlargement. The *Strongyloides stercoralis* (see above, Fig. 214, page 401) has also been found in the urine.

1. *Cystitis.*—Urine passed frequently, painfully, and in small amounts. Turbid, ammoniacal, and offensive (after the earliest stages). Much pus and many other cells are found in the sediment, with bacteria, triple phosphate crystals, and amorphous débris. It must be remembered that cystitis is usually but one element in the diagnosis; bladder stone, obstructing prostate, tuberculous kidney, or other diseases may be its cause.

2. *Acute Nephritis* (or acute exacerbations in chronic cases).—Scanty, heavy, highly albuminous urine, often bloody and containing

in the sediment *much blood* and many cells, free or on casts. Other varieties of casts occur, but are not characteristic. In convalescence the urine becomes abundant and of light weight, and the other abnormalities gradually disappear.

3. *Chronic Nephritis* ("parenchymatous" or "diffuse").—The urine is rather scanty, pale, and light (1.012–1.018), with a varying amount of albumin and, in the sediment, *much fat*— free, in cells, and on casts. Also found, but not characteristic, are all the other varieties of casts. If death does not ensue within eighteen months, the urine is apt to assume the different characteristics:

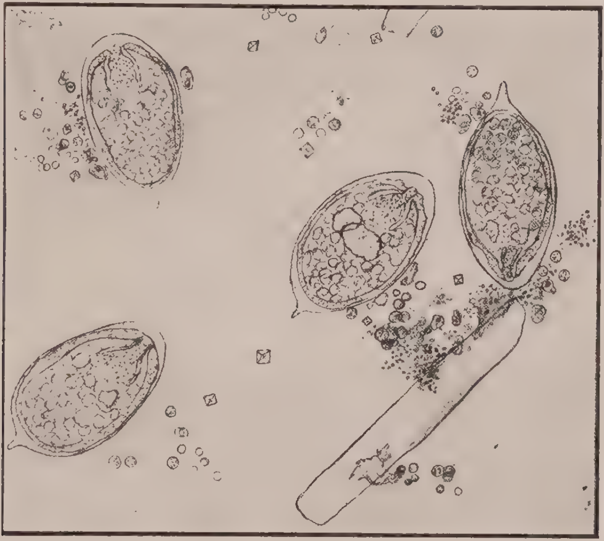

FIG. 234.—Bilharzia Eggs in the Urine, with Blood, Calcic Oxalate, and a Hyaline Cast. (O'Neil.)

4. The later stages of chronic nephritis show often polyuria (often several quarts; urine especially abundant at night), low specific gravity (1.014 or less). Traces of albumin and a few hyaline and granular casts occur steadily or intermittently. High blood pressure is nearly constant.

5. *Pyelitis and acute hæmatogenous renal suppurations* are diseases much more commonly recognized since 1904 than previously. The presence of bacteria (usually colon bacilli) and pus without many cells of other types in acid urine should always lead to bacteriological

and cystoscopic examinations. This condition of the urine may at times be the only sign of the disease. The presence of pain, tenderness, or tumor in the region of the kidney (usually the *right* kidney) and the occurrence of fever and leucocytosis support the diagnosis, especially in little girls and in women near parturition. To distinguish *pure pyelitis from pyelitis* complicating a renal infection is impossible.

This disease should be borne in mind:

(*a*) In all cases of unexplained fever without obvious local cause—especially in girl babies and in women a short time before or after parturition (subacute or chronic renal infection).

(*b*) In acute abdominal emergencies when appendicitis, cholecystitis, intestinal obstruction, perforating peptic ulcer, and pancreatitis are being considered. *With these consider also acute infection* of the kidney, for in some cases the pain is in the right hypochondrium and no complaints suggesting the kidney are uttered.

CHAPTER XXIV

THE BLADDER, RECTUM, AND GENITAL ORGANS

I. The Bladder

1. *Incidence of Bladder Disease*

(Massachusetts General Hospital, 1870–1905.)

Cystitis	829 cases.
Stone	538 cases.
Cancer	57 cases.
Papilloma	20 cases.
Tuberculosis	43 cases.

(*a*) *Data*

Distention, *tumor*, *the urine*, and the results obtained by *cystoscopy*, by *catheterization*, by *rectal* and *vaginal* examination, and by *the x-ray*, furnish most of our direct evidence in bladder disease. Pain in the bladder or near the end of the penis, and frequent, painful micturition with vesical tenesmus or straining, are common symptoms in various lesions of the organ, and direct our attention to it, though they do not indicate the nature of the trouble.

2. *Distention of the Bladder*

In both sexes, distention is often wholly unknown to the patient, and may be accompanied by frequent acts of urination, especially in prostatic obstruction, in acute infections, and after operations. A distended bladder is readily recognized by palpation as a smooth, round, firm, symmetrical tumor in the median line, above the pubes. The tumor is *dull on percussion*, and in slight degrees of distention this *dulness above the pubes* may be the only physical sign obtainable. In well-marked cases, which are most common in males, the distended bladder may reach to the navel or even above it, and the beginner is usually astonished at its dimensions and its firm, resistant surface (see

Fig. 235). Diagnosis rests on the infrequency of other tumors of this region in men and on the result of catheterization or suprapubic aspiration. In females a history of failure to pass urine almost invariably makes the diagnosis obvious, though occasionally after operations distention of the bladder and dribbling of urine may go together in women, as they so frequently do in men.

The commonest causes of distended bladder are:

(1) Prostatic obstruction.

(2) Old strictures of the urethra.

Less common are:

(3) Spasm of the urethra in gonorrhœa.

(4) Acute prostatitis.

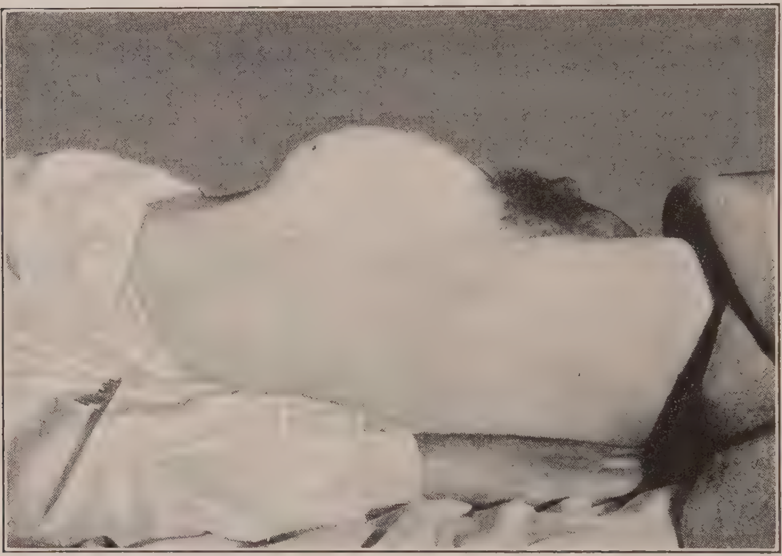

FIG. 235.—Distended Bladder Reaching Above the Navel.

(5) Paralysis of the bladder, from disease or injury, after operation, and in fevers.

(6) Tumor or stone near the neck of the bladder.

The diagnosis of the cause of distention rests on the history, the result of attempts at catheterization, the rectal examination, the condition of the urine, and the physical signs in other parts of the body. A long history of frequent micturition, especially at night, in an old man, an obvious enlargement of the prostate felt by rectum, and the passage of ammoniacal urine suggest *prostatic obstruction.* The information obtained during the passage of a catheter usually clinches the diagnosis.

Acute retention, with no previous history of frequent micturition or foul-smelling urine in a young or middle-aged man, who has had gonorrhœa and may or may not have noticed a diminution in the size of the stream of urine passed, suggests a *urethral stricture.* The catheter decides.

Spasm of the urethra may occur in acute gonorrhœa, and produces a retention which may often be overcome by hot poultices and enemata. The history and the effects of treatment suggest the cause of the retention.

Acute prostatitis, as a cause of retention following gonorrhœa, is suggested by pain and tenderness in the perineum, *painful defecation*, fever, perhaps chills, and a hot, tender prostate felt by rectum. Abscess may form and discharge by urethra or rectum.

Paralysis of the bladder, as a cause of retention, is usually obvious from the history and from the evidence of disease of the spinal cord, or of operation and semicomatose states (as in fevers and shock).

3. *The Urine as Evidence of Bladder Disease*

This has been described above (page 427). Cystitis, acute or chronic, usually gives characteristic evidence of itself in the urine, and suggests as its cause the possibility of gonorrhœa, of vesical stone, of prostatic or other obstruction to the outflow, and of vesical or renal tuberculosis. When a urine like that described in chronic nephritis occurs with chronic prostatic obstruction the relief of the obstruction is necessary if we are to prevent progressive development of cirrhotic kidney from back pressure.

Frequent micturition is much commoner and less significant in women than in men. All sorts of "nervousness" and emotional strain produce this symptom in women, independent of any demonstrable source of irritation in the urinary tract. Aside from these conditions the symptom is oftenest met with in:

(*a*) *Cystitis*, from any cause, including stone and renal tuberculosis, or without known cause, with characteristic changes in the urine.

(*b*) *Prostatic obstruction*, with evidence of retention.

(*c*) *Gonorrhœa*, with evidence of this disease.

(*d*) *Paralysis of the bladder* (see above).

(*e*) *Overconcentration of the urine* (estimated by the color and specific gravity).

III. Stone in the Bladder.—Pain near the end of the penis, especially at the end of micturition and aggravated by jolting or active motion, frequent urination, especially in the daytime, sudden interruption of the stream of urine, and hæmaturia at the end of micturition, are the most frequent symptoms of stone, especially if they occur in boys. In old men stone may be wholly without characteristic symptoms, and at any age the symptoms can never do more than suggest the possibility of stone and the advisability of searching for it systematically with a cystoscope.

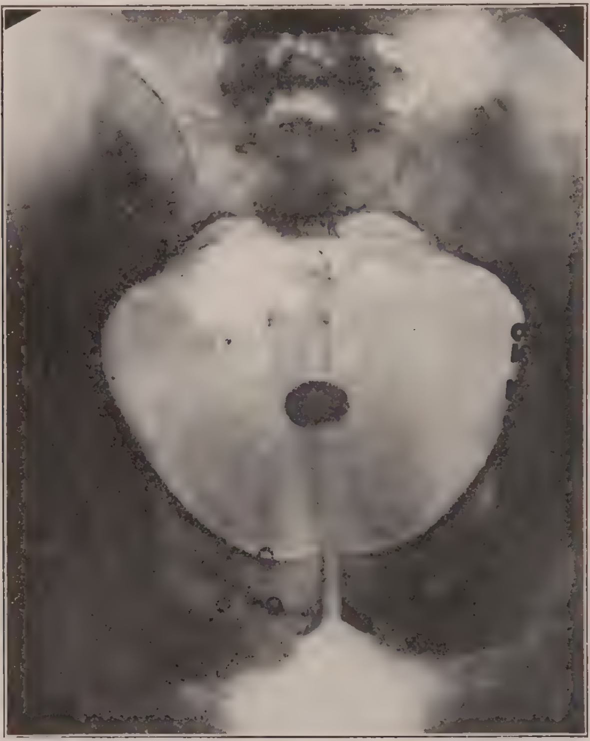

FIG. 236.—X-ray of stone in the bladder.

IV. Tuberculosis of the Bladder.—Cystoscopy and the recognition of tubercle bacilli by animal inoculation are the only reliable means of diagnosis. A chronic cystitis in a young or middle-aged person, especially with an acid urine, is suggestive.

V. Tumors of the bladder are suggested by intermittent hæmaturia with vesical irritation, and confirmed by cystoscopic examination.

II. The Rectum

1. *Symptoms*

It is not and should not be a part of routine physical examination to examine the rectum. The commonest conditions which call for such investigation are:

(*a*) Hemorrhage at stool.

(*b*) The protrusion after defecation of something which is not easily returned ("piles").

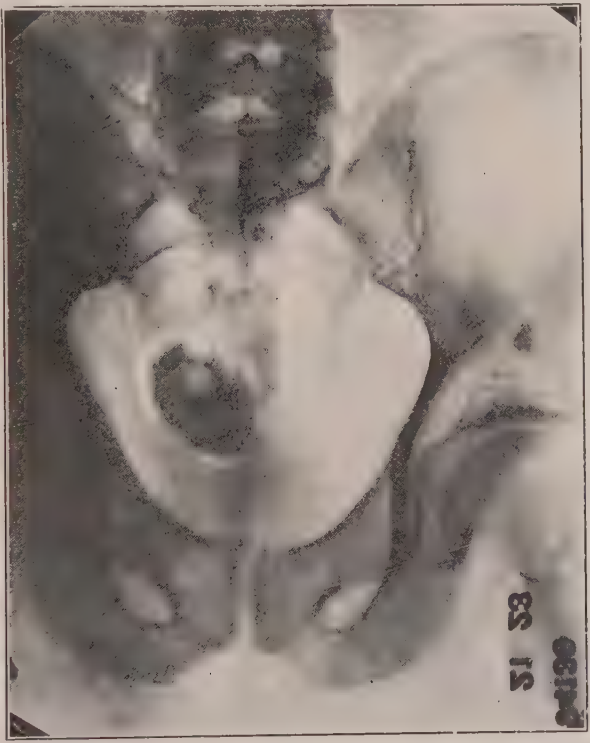

Fig. 237.—X-ray of stone in the bladder.

(*c*) Painful defecation or pain in the region of the rectum at other times.

(*d*) The presence of an ulcer or sinus near the rectum.

(*e*) Habitual constipation, not explained by lesions elsewhere.

(*f*) Intestinal obstruction.

(*g*) All subacute diarrhœas of elderly persons (cancer).

(*h*) Suspected appendicitis, prostatitis, prostatic cancer or obstruction, or diseases of the seminal vesicles.

(*i*) Pelvic symptoms in women with tight hymen.

The diseases of the rectum which we are especially on the lookout for are: (1) Hemorrhoids; (2) fissure of the anus; (3) ischiorectal abscess; (4) fistula in ano; (5) cancer of the rectum. Less common are: (6) pruritus ani; (7) prolapse of the rectum; (8) ulceration or stricture of the rectum.

2. *Methods*

For most examinations the finger suffices. It should be covered by a thin, rubber finger-cot, greased with vaseline, and should be introduced slowly and gently while the patient strains down as during defecation.

The examining finger should note the presence of abnormal prominences or resistance (piles, tumors) in any part of the rectum, of tender spots (ulcer, abscess), and strictures. The shape and size of the prostate gland, its consistence, and the presence or absence of tenderness in it are of importance. The normal seminal vesicles can be felt if distended. If they are hard and nodular, tuberculosis should be suspected. By milking the vesicles and massaging the prostate, pus containing gonococci may be pressed out through the urethra. This process is often of value both in diagnosis and in treatment.

High up on the right one may touch a tender spot if an inflamed appendix is near the pelvic brim (often a fallacious sign).

In women the uterus, especially if retroverted, may be easily felt, and most of the other details of pelvic examination (see below, page 440), can be more or less clearly made out.

For higher and more thorough examination a cylindrical speculum and a head mirror should be used.

Hemorrhoids.—The diagnosis of *external hemorrhoids*, which can easily be brought outside the anus, is made at a glance. *Internal hemorrhoids* are best seen with a rectal speculum, and may resemble the external or may consist of "bright red, spongy, granular tumors, rarely larger than a ten-cent piece, and situated high up in the rectum."

Fissure of the anus is often connected with a small ulcer and with œdematous folds, which resemble an external pile but are much more tender. On separating these folds the fissure comes into sight. It usually produces severe pain during and after defecation.

Ischio-rectal abscess presents near the anus the ordinary signs of abscess with pain radiating through the pelvis, but may open either within or outside the rectum and results in

Fistula in ano, a sinus beside the rectum, opening internally, externally, or in both directions. It may be very tortuous. Tuberculosis is to be suspected in such fistulæ.

Cancer of the rectum is suggested by the occurrence of rectal pain during defecation, with blood in the stools and either diarrhœa or constipation, usually with some pallor and emaciation, in persons past middle life. Owing to neglect of a thorough examination many cases are at first mistaken for piles.

The examining finger reaches a hard, ulcerating mass high up, as a rule, in the rectum. It may be easier to reach if the patient stands or squats and strains down during examination.

From tuberculous or syphilitic stricture with or without ulceration, and from benign villous growths, it may be impossible to distinguish cancer without histological examination of an excised piece. Cancer of the prostate is felt on the anterior wall of the rectum and makes the prostate hard, fixed, sometimes nodular. It is often very difficult to recognize.

III. The Male Genitals

Routine examination of the male genitals includes investigation of the penis for the presence of:

(*a*) Urethral discharge and its consequences.
(*b*) Chancre.
(*c*) Chancroid.
(*d*) Balanitis.
(*e*) Phimosis or paraphimosis.
(*f*) Periurethral abscess.
(*g*) Malformations.
(*h*) Cancer.

In the testes and scrotum we look for:

(*a*) Epididymitis (gonorrhœal or tuberculous).
(*b*) Orchitis (traumatic, syphilitic, tuberculous, after mumps and other infections).
(*c*) Tumors of the testis (cancer or sarcoma).
(*d*) Hydrocele and hæmatocele.
(*e*) Varicocele.
(*f*) Scrotal hernia.
(*g*) Absence of one or both testes.

1. *The Penis*

Urethral discharge, if not obvious, may often be brought to light by "stripping" the urethra forward from the prostatic region to the meatus. If Gram's stain brings out an intracellular, decolorizing diplococcus in the exudate, there is no reasonable doubt of the presence of gonorrhœa.

Chancre ("hard sore"), the primary syphilitic lesion, is a superficial, painless, indolent ulcer with an *indurated base* and a scanty serous discharge. It is usually round or oval and sharply demarked from the surrounding tissue by elevated edges. It is rarely multiple. Painless, hard, non-suppurating buboes accompany it. The glans and the inner surface of the prepuce are the commonest sites. The Treponema pallidum can often be identified in stained smears or by the dark field illumination. In a certain percentage of cases a positive Wassermann reaction may be obtained.

Chancroid ("soft sore") is like any other painful, superficial ulcer without induration, irregular in shape, often multiple, and with abundant discharge. A single, painful bubo accompanies it in about one-third of all cases.

Balanitis (inflammation of the surface of the glans penis), usually gonorrhœal, has the ordinary signs of inflammation; it often spreads to the inner surface of the prepuce.

Phimosis is a contraction of the orifice of the prepuce, so that it cannot be retracted to uncover the glans. May be hereditary or the result of gonorrhœa.

In *paraphimosis* the prepuce is caught behind the glans penis so that it cannot be brought forward. Great œdema of the neighboring parts usually results.

Peri-urethral abscess, usually a complication of gonorrhœa, appears as a small, tender swelling on the under surface of the urethra.

Malformations are chiefly *hypospadias* or congenital deficiency of some portion of the lower wall of the urethra, and *epispadias* (rare), a similar deficiency in the upper wall. A short, downward curved penis is often associated with hypospadias.

Cancer of the penis attacks the foreskin or the glans, and has the usual characteristics of epithelioma elsewhere.

2. *The Testes and Scrotum*

Acute epididymitis, usually a complication of gonorrhœa, appears as a hot and tender swelling behind the testis, often preceded by tenderness along the spermatic cord. Acute hydrocele may accompany it.

Chronic epididymitis, usually *tuberculous*, is painless and insidious in onset, and produces a hard, irregular enlargement low down behind one or both testes, to which, however, the process is apt soon to spread. Caseation and involvement of the skin later produce a suppurating sinus, which is often the first thing to bring the patient to a physician.

Acute orchitis is often due to a blow, to gonorrhœa, or to mumps. The testis is symmetrically swollen and tender, but suppuration rarely follows.

Chronic orchitis, often *syphilitic*, is slow, painless, and may be accidentally discovered as a slightly irregular induration of the testes with little if any increase in size. Ulceration and fistulæ are rare in the syphilitic form, common in the tuberculous.

Cancer of the testis may appear at any age. It is soft, almost fluctuating, and grows very rapidly, soon involving and perforating the skin, so as to produce an offensive, fungous, granulating outgrowth which easily bleeds. The inguinal glands are involved.

Sarcoma of the testis, commonest at puberty, produces a painless, uniform enlargement, and may reach great size. It may resemble hydrocele or hæmatocele and be mistaken for the latter, especially for an old effusion in a thickened sac (see below).

Diagnosis depends on rapid growth, the *entire* absence of translucency, the tendency to adhere to the skin and to present unequal resistance in different portions (Jacobson). Incision should be made in all doubtful cases.

Hydrocele, an accumulation of serous fluid in the tunica vaginalis, may depend on trauma or on an acute epididymitis or orchitis, but is usually chronic and of unknown cause. It may be congenital and communicate with the peritoneal cavity or form part of a general dropsy in heart or kidney disease.

Examination shows a smooth, tense, fluctuating tumor, without impulse on cough, usually without pain, tenderness, or any sign of inflammation, and, above all, *translucent* if examined with a hydroscope tube or in a dark room with a candle.

If the fluid is opaque or bloody, or if the tunica is thickened, there may be no translucency and diagnosis may be impossible without puncture. The *testis lies behind* the effusion and near its lower end.

Hæmatocele usually follows injury and produces a heavy, opaque, non-fluctuating tumor, which may closely resemble sarcoma unless the history and evidence of trauma are clear. Incision or puncture should decide.

Varicocele, a usually harmless enlargement of the veins about the spermatic cord, is easily recognized as a mass of tortuous vessels, generally in the left side. It often complicates hypernephroma.

Scrotal hernia is usually reducible, tympanitic on percussion and gives an impulse on coughing. If it consists largely of omentum it will be dull on percussion. The history of the case and the progression of the tumor from above downward usually make its origin clear.

Absence of one or both testes from the scrotum should direct our search upward to the inguinal canal, since a retained testis may be the seat of troublesome inflammation or of malignant disease. (For examination of the seminal vesicles, see the Rectum, page 436.)

IV. The Female Genitals

1. *Methods*

Inspection of the external genitals is easy if the parts are properly exposed by a satisfactory position and a good light. Intravaginal inspection needs a speculum (Sims' or bivalve) and usually an assistant to hold it.

Palpation should always be bimanual, the left forefinger in the vagina (or in the rectum if the hymen is narrow), the right hand above the symphysis pubis. The proper co-operation of the hands is hard to describe and depends on practice. The pressure of the external hand helps to bring the pelvic organs within reach of the examining finger in the vagina. Unless the organs can be thus grasped or balanced between the outer and inner hands, no satisfactory examination is possible. Tenderness may prevent this or render an anæsthetic necessary, but gentleness and the avoidance of any sudden or rapid motions do much to facilitate the examination. The left hand, in making its way into the upper parts of the vaginal vault, should press only on the perineum, avoiding the region of the clitoris. It is astonishing how much pressure can be borne without pain, provided it is exerted gradually and upon the perineum only. Many examiners find it advantageous to rest the left foot upon a stool, with the left elbow on the knee.

2. *Lesions*

I. In the External Genitals one looks for some of the same lesions already described on page 438, viz., chancre, chancroid, local inflammations, and tumors. Only the commonest and most important lesions will be mentioned here.

(*a*) In young children a suppurating *vulvo-vaginitis*, usually gonorrhœal, but often non-venereal, is easily recognized by the abundant purulent discharge.

(*b*) *Local eczema*, often red and angry, is commonly the result of the irritation of *diabetic urine* or a leucorrhœal discharge.

(*c*) *Varicose veins* and œdema of the vulva are common in pregnancy and occasionally result from large pelvic tumors.

(*d*) *Ruptured perineum*, with more or less protrusion of the vaginal walls, carrying with it the bladder (cystocele or rectum (rectocele), is readily recognized if the normal anatomy of the parts is familiar.

(*e*) *The hymen* may be *imperforate* with retention of menstrual fluid, or tender, irritated remains of it after rupture may cause pain and need removal.

(*f*) *Urethral caruncle* (a small vascular papilloma at the entrance of the urethra) is a bright red excrescence, usually the size of a split pea or smaller. It may cause no symptoms or may produce irritation, especially during micturition.

(*g*) *Small abscesses* of the glands within or around the urethra may cause pain in coitus or during micturition.

II. THE UTERUS.—Only the commonest lesions will be dealt with here, viz.:

1. Laceration and "erosion" of the cervix.
2. Malpositions of the organ.
3. Endometritis.
4. Cancer of the uterus.
5. Fibor-myoma of the uterus.

1. (*a*) *Lacerations of the cervix* following childbirth are very common and frequently produce no symptoms. They are readily recognized by inspection and palpation, and are often combined with:

(*b*) "*Erosions*," an ulcerated, raw surface at and around the os uteri with or without the formation of small cysts. At times the os assumes a warty, irregular appearance, suggesting cancer, from which it can be distinguished only by histological examination of an excised piece.

2. (*a*) "*Malpositions*" (backward or forward) may involve the whole organ (ante- or retroversion) or represent a bending of the organ upon itself (ante- or retroflexion). These lesions may be variously combined and frequently exist without producing any symptoms. Indeed, it is doubtful whether there is any single "normal"

position for the uterus. Its position is recognized by bimanual palpation, which should also determine whether the uterus is freely movable or whether it is bound in place by adhesions, such as are very often found with backward displacements.

(*b*) *Prolapse of the uterus* toward the vaginal outlet is often a result of pelvic lacerations unrepaired. When the uterus is outside the vaginal outlet, we call the condition *procidentia.*

(*c*) *Lateral displacement* of the uterus by pressure of tumors or traction by old adhesions is less common.

3. *Endometritis* may present no definite physical signs except a muco-purulent discharge (leucorrhœa, "whites") and perhaps unduly frequent, profuse, or prolonged menstruation. The slightest touch of a uterine sound may produce bleeding. It often accompanies disturbances of digestion and neurasthenic conditions, usually as part of a general prostration.

4. *Cancer of the uterus* usually attacks the cervix, and in marked cases is easily recognized by sight and touch as a "cauliflower"-like, *fungating mass* on the cervix. In its early stages it may be confounded with "erosions" and papillomatous growths, and only microscopic examination can satisfactorily determine its nature. *Profuse hemorrhage*, especially in a woman about the period of the menopause, and the *offensive* odor of the *discharge* suggest the diagnosis. The vaginal wall is soon involved in the growth, and irritability or obstruction in bladder or rectum may result. Cancer of the fundus is suspected from the finding of enlargement or a suspicious discharge, but confirmed only by the histological examination of bits removed by curetting.

5. *Fibro-myoma of the uterus* is by far the commonest tumor of that organ. It produces hemorrhages at or between the menstrual periods, and anæmia results. Otherwise its effects are those of pressure on the bladder and rectum, or on neighboring nerves or vessels (pain, œdema).

Bimanual palpation determines, first of all, the fact that the growth is connected and moves with the uterus. This determined and cancer excluded by the absence of any involvement of the cervix or of the vaginal wall, the chief difficulty may be in distinguishing the growth from a pregnant uterus. Usually its irregular shape, the persistence of menses, and the lapse of time settle the question.

Lengthening of the uterine canal is an important confirmatory sign of fibromyoma, but sounds should never be passed to determine this fact unless pregnancy is definitely impossible.

III. Fallopian Tubes.—Salpingitis (acute or chronic) and tubal pregnancy are the most important diseases of the tubes.

(*a*) *Salpingitis* is usually gonorrhœal, occasionally tuberculous, sometimes of unknown origin. A painful, tender swelling or induration in the region of the tube, with or without fever, chill, or leucocytosis, constitutes the evidence for diagnosis. From pelvic peritonitis of the tubal region diagnosis is impossible.

From tubal pregnancy diagnosis may be very difficult, and suspicions are rarely aroused until rupture occurs (*vide infra*). If the signs and symptoms of pregnancy are absent and tenderness is marked the condition is usually called salpingitis; but even then mistakes often occur, as the menses may persist in tubal pregnancy and the fœtal tumor may be tender. Only when pregnancy can absolutely be excluded is diagnosis sure.

(*b*) *Tubal pregnancy*, as just explained, is rarely to be diagnosed until the growth of the fœtus ruptures the tube—an event which usually occurs between the third and the twelfth week of pregnancy.[1] Sudden pelvic pain with tenderness, vomiting, and evidence of internal hemorrhage (*i.e.*, pallor, fainting, weak, rapid pulse, thirst, air hunger) suggest the diagnosis, especially if a tumor in the tubal region can be detected bimanually.

IV. Ovaries.—A *prolapsed* ovary is often felt during a vaginal examination, being recognized by its size, shape, and relation to the uterus.

Ovaritis, enlargement, and tenderness of one or both ovaries is usually part of tubal disease and not sharply to be distinguished from it before operation. In other cases it is associated with *cyst formation*, and the cysts may be palpated bimanually. *Abscess* of the ovary is not commonly diagnosed, but is met with in operations for pus tubes.

Ovarian Tumors

(*a*) *Small Tumor.*—In their earlier stages these growths produce symptoms only when complications arise, *i.e.*, suppuration or twisting of the pedicle. *Small, suppurating cysts* give practically the same signs as those of a pus tube, and are recognized only at operation or autopsy.

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[1] If disturbances of menstruation, morning nausea, changes in the breasts, and cyanosis of the vagina are combined with an extra-uterine tumor and an unusually slight uterine enlargement, the diagnosis of tubal gestation may be *suspected* prior to rupture.

Twisted pedicle gives rise to symptoms and signs often indistinguishable from those of perforative peritonitis or intestinal obstruction. Only the recognition of the tumor as ovarian can suggest that the acute symptoms may be due to twisting of its pedicle.

(*b*) *Large ovarian tumors* have been confused in my experience with pregnancy, fibroid of the uterus, ascites, and tuberculous peritonitis. From these we may usually distinguish an ovarian tumor by its history, its origin from one side of the belly, by the shape of the belly, the area of percussion dulness, and the pelvic examination.

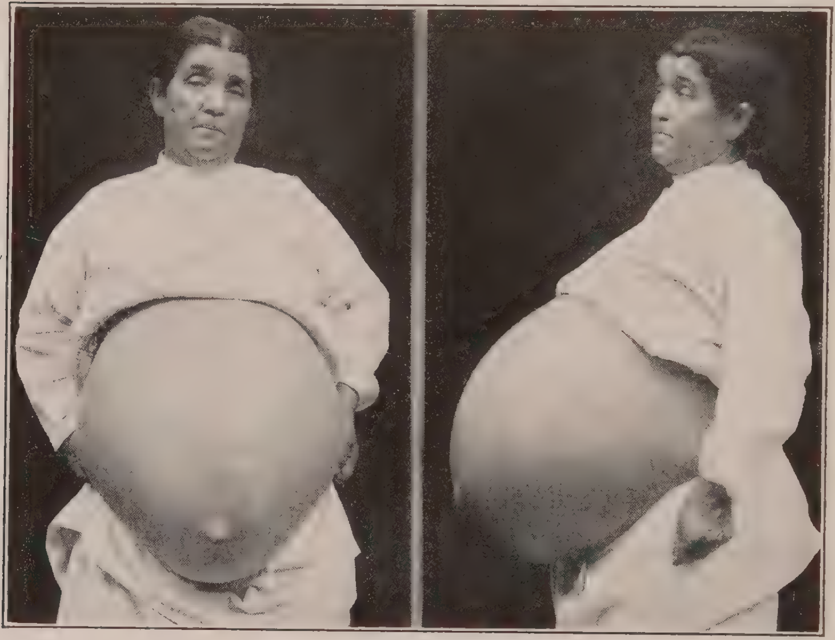

FIG. 238.—Huge Ovarian Cyst.

By the history we should attempt to exclude disease of the heart, kidney, and liver, and tuberculosis of any organ, should inquire into the position of the tumor in the earlier stages of its growth, and establish the presence or absence of the ordinary signs of pregnancy and of uterine hemorrhages such as occur with fibroids.

In ascites or tuberculous peritonitis the flanks often bulge (see Fig. 204, page 364), whereas in ovarian disease the bulging is central and greatest just below the navel (see Fig. 238).

If by the history or by palpation and percussion we can determine that the tumor is fluctuant and springs from one side of the abdomen, it is in all probability ovarian. High psoas abscess sometimes presents identical signs, but is associated with evidence of spinal tuberculosis (see below, p. 482). *Moderate* ascites or tuberculous peritonitis leaves an oval, resonant area about the navel, which is absent with large ovarian tumors; but if the amount of free fluid is large, percussion and palpation may give results identical with those found in ovarian disease.

Vaginal examination may exclude fibroid by showing that the uterus is not directly connected with the tumor and by demonstrating with a uterine sound that the uterine canal is not elongated.

Solid tumors of the ovary, carcinoma, sarcoma, or fibroma are rarely recognizable before operation and are often mistaken for pedunculated uterine fibroids. They are apt to be associated with ascites.

CHAPTER XXV

THE LEGS AND FEET

I. The Legs

1. *Hips*

The examination of the hip will be discussed later (see page 482).

2. *Groin*

In the groin we look for evidences of:

1. Enlarged or inflamed lymphatic glands and scars of previous inflammation.
2. Hernia and hydrocele of the cord.
3. Psoas abscess.

Less common are:

4. Retained testis.
5. Filarial lymphatic varix.

1. *Inguinal Glands.*—Two sets of inguinal glands are distinguished—one arranged along the lower half of Poupart's ligament; the other lower down, around the saphenous opening.

(*a*) The "Poupart's group" are acutely enlarged in lesions of the genitals ("bubo" or gonorrhœa,[1] syphilis, chancroid) and perineum; chronically enlarged in malignant disease of the penis, uterus (late), and other genitalia.

(*b*) The saphenous group is enlarged in response to lesions of the thigh, leg, and foot (cuts, wounds, ulcers, eczema, etc.).

(*c*) Either or both groups may be enlarged in leukæmia, Hodgkin's disease (see above, page 32), infectious arthritis, and various obscure fevers. In many cases no cause for enlargement can be found.

2. *Hernia* is diagnosed by the presence of a soft, resonant, fluctuating, usually reducible tumor with an impulse on coughing. *Hydrocele of the cord* gives also an impulse on coughing, but usually shows a distinct limit above. On pulling the cord the swelling moves too.

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[1] The bubo of gonorrhœa often suppurates; that of syphilis rarely. Hence a scar in the inguinal region suggests an old gonorrhœa.

3. *Psoas abscess* (see Fig. 239) is associated with vertebral tuberculosis. Don't cut into it without excluding aneurism!

4. *Retained testis* should be suspected whenever an inguinal tumor is present and only one testis is found in the scrotum.

5. *Filarial lymphangiectasis* is generally mistaken for hernia and operated on as such, although it gives no impulse on coughing and cannot be completely reduced. The history of residence in the tropics should always suggest an examination of the blood (at night) for filariæ.

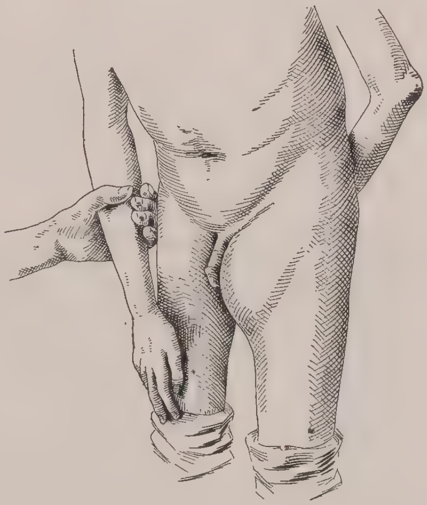

FIG. 239.—Psoas Abscess. (Bradford and Lovett.)

3. *The Thigh*

The records of the Massachusetts General Hospital show that (1) *epiphysitis* and *osteomyelitis* (septic or tuberculous) are almost ten times as common as any other serious lesion of the thigh, except fracture. The cases are to be divided into acute septic cases and chronic, usually tuberculous, cases.

The acute septic cases begin with severe pain, tenderness, fever, chill, and leucocytosis. Later an induration and finally fluctuation appear, and the abscess, if not incised, will break externally. General, sometimes fatal, septicæmia may take place.

The chronic tuberculous cases first consult the physician, as a rule, for *sinus*, which proves when explored to lead to dead bone, as do most of the sinuses from septic cases.

The diagnosis of the acute cases depends chiefly on excluding arthritis of any type. Careful examination with testing of joint motions will usually demonstrate that the pain and tenderness are in the bone and not in the joint. The leucocyte count is but slightly elevated in most cases of arthritis, but is decidedly high, 20,000 or more, in most cases of acute osteomyelitis. The same is true of the temperature. Monarticular arthritis—the only variety likely to be considered in such a diagnosis—is rare in youth, when most cases of acute osteomyelitis and epiphysitis occur.

Whether the disease starts in the shaft of the bone or in the epiphysis is to be determined by the seat of pain and tenderness.

Tuberculous cases can be recognized only by the histological examination. Old cases may be suspected by the presence of a scar, but

(2) *Multiple white scars* should always suggest, though they are far from proving, syphilis, for *chronic ulcer* above the knee is often due to gumma.

(*a*) *Tumors of the Thigh*

(1) *Sarcoma of the femur* is the commonest and largest tumor of the thigh. Among one hundred and thirty-three tumors of the thigh recorded at the Massachusetts General Hospital, sixty-six were sarcoma. A hard, spindle-shaped growth encircles the femur; the lower end is the commonest site, but any part of the bone may be affected (see Fig. 240).

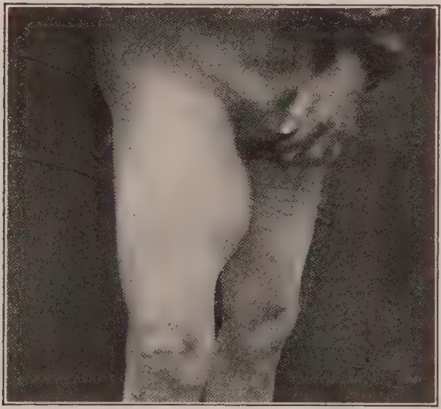

FIG. 240.—Sarcoma of the Femur.

(2) *Osteoma*, or exostosis, occurred eleven times in the one hundred and thirty-three cases just mentioned. It is much smaller and of slower growth. The last trait usually serves to distinguish it from sarcoma; *x*-ray should decide.

(3) *Metastatic cancer* of the upper half of the femur may occur after cancer of the breast, but rarely gives rise to symptoms unless spontaneous fracture occurs—an event which always should suggest cancer. Epithelioma of the thigh is not very rare (twelve cases in the one hundred and thirty-three above referred to). Its traits are those of epithelioma elsewhere.

Tuberculosis of the knee may simulate sarcoma of the lower end of the femur, but sarcoma grows more rapidly. The tuberculin test or an exploratory incision may be necessary to decide the diagnosis.

(4) *Psoas abscess* or *hip-joint abscess* (see Fig. 239) may burrow down so as to point on the thigh. The evidence of disease in the hip or vertebræ is usually sufficient to make clear the diagnosis.

1. *Sciatic pain* and tenderness more or less clearly confined to the distribution of the sciatic nerve may be due to

(*a*) Sacroiliac disease (strain, looseness, displacement).
(*b*) Spondylitis.
(*c*) Unknown cause (neuritis).
(*d*) Prostatitis, prostatic abscess or neoplasm.
(*e*) Pelvic tumors or abscess (including psoas abscess).

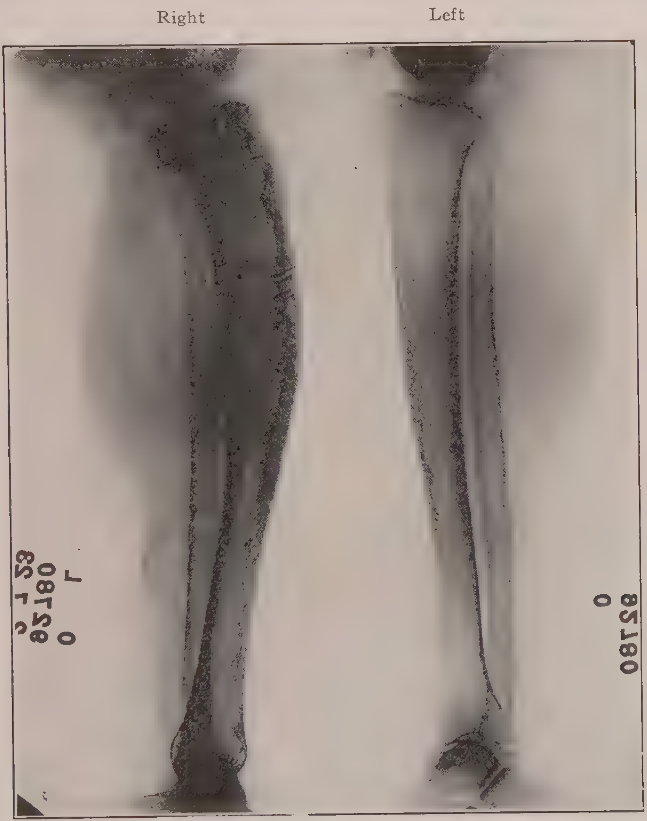

FIG. 241.—Paget's disease. X-ray of tibiæ. Right much more extensively diseased.

(*b*) *Miscellaneous Lesions of the Thigh*

(2) *Phlebitis* with thrombosis of a vein, usually the saphenous, is a common cause for swollen thigh (and leg) with pain and tenderness, especially over the inflamed vein, where a cordy induration can often be felt. Typhoid fever and the puerperal state are the usual

causes, but it also occurs after tonsillitis and other infections, and sometimes without any known cause. Diagnosis depends on the presence of these signs and causes and the absence of any other demonstrable cause for inflammation.

(3) *Meralgia parœsthetica* means the presence of a patch of anæsthesia, paræsthesia, or hyperæsthesia (tenderness), with or without pain, on the anterior and upper surface of both thighs (the area of the external cutaneous nerve).

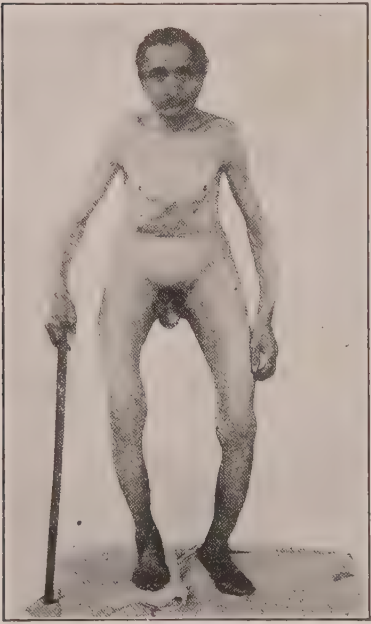

FIG. 242.—Paget's Disease (Osteitis Deformans). Note the outward and forward bowing of legs and arms. (Robin.)

(4) *Paget's disease* (osteitis deformans) presents usually its most marked lesions in the legs and head, though most of the other bones are also affected. In the leg the most characteristic lesions are forward bowing of the femur and tibia with outward rotation of the whole limb (see Fig. 242). The *x*-ray shows marked thickening of some areas, with thinning of others.

(5) *Intermittent Claudication and "Cramps."* Insufficient circulation through the arteries of the legs may give rise to sudden "giving way" of one or both during running or walking, the power returning after a short rest. In patients at rest the frequent recurrence of painful cramps in the muscles may be the only manifestation of the disease. In other cases there are various forms of paræsthesia such as numbness, prickling, and "hot feet at night."

Obliteration of the dorsalis pedis (or larger arteries) by arteriosclerosis is often found, but there is reason to believe that local anæmia, due to vasomotor disturbances or other causes, may produce similar cramps (*e.g.*, those seen in football players during a hard run and in pregnant women).

(*c*) *Paralyses*

(1) *Paralysis of one leg*, occurring in children, is usually due to *anterior poliomyelitis;* in adults it usually forms part of a *hemiplegia* or

is of *hysterical* origin. *Neuritis*, due to alcohol, lead, arsenic, or diphtheria, may affect one leg predominantly, but both are usually involved. *Cerebral monoplegias*, due to cortical lesions of the leg area, are rare. *Chorea* may be associated with a limp, half-paralyzed condition in one leg, usually with some involvement of the arm on the same side, and the characteristic motions (see above, page 48) make the diagnosis clear.

The differential diagnosis of the other varieties of monoplegia is usually easily made with the aid of a careful history and a thorough examination of the other parts of the body.

(2) *Complete paralysis of both legs* (paraplegia) is commonest in diffuse or transverse myelitis (*e.g.*, in spinal tuberculosis or metastatic cancer with pressure on the cord), in multiple sclerosis; spastic paraplegia (hereditary or acquired), pernicious anemia and tabes. Hysteria also may produce a spastic paraplegia, though monoplegia is commoner in this disease.

(3) *Partial paralysis of both legs* is oftenest due to neuritis, resulting from the causes mentioned above. The extensors of the foot are especially affected and toe-drop results, so that in walking "the entire foot is slapped upon the ground like a flail" (Osler).

Differential Diagnosis.—(*a*) In *diffuse or transverse myelitis*, whether or not the trouble be due to pressure, there are increased reflexes, anæsthesia, usually loss of control of the sphincters (involuntary urine and fæces), and often bed-sores.

(*b*) In *spastic paraplegia* of any type the legs are stiff and the reflexes increased, but sensation and the sphincters are normal and there is no atrophy or bed-sore formation.

(*c*) In *multiple sclerosis* there are usually no disturbances of sensation or of the sphincters, and the paralysis is associated with nystagmus, intention tremor, and slow, staccato speech.

(*d*) *Tabes dorsalis* shows *ataxia* but no paralysis until late in its course. The paralytic stage is preceded by a long period characterized by lightning pains, bladder symptoms, Argyll-Robertson pupil (see page 16), and loss of knee-jerks.

(*e*) *Hysteria* may take on almost any type of paralysis and may deceive the very elect, but as a rule the other evidences of hysteria guide the diagnosis.

4. *The Knee*

(*a*) Tuberculosis, atrophic, hypertrophic, and infectious arthritis, and traumatic synovitis are the commonest diseases, but will be described with other diseases of the joints (see page 480).

(*b*) Housemaid's knee is a bursitis of the prepatellar bursa (see Fig. 243). Fluctuation, with or without heat and tenderness, and limited to the prepatellar space, is diagnostic.

(*c*) Bow-legs and knock-knee are so easy of diagnosis that I shall simply mention them here.

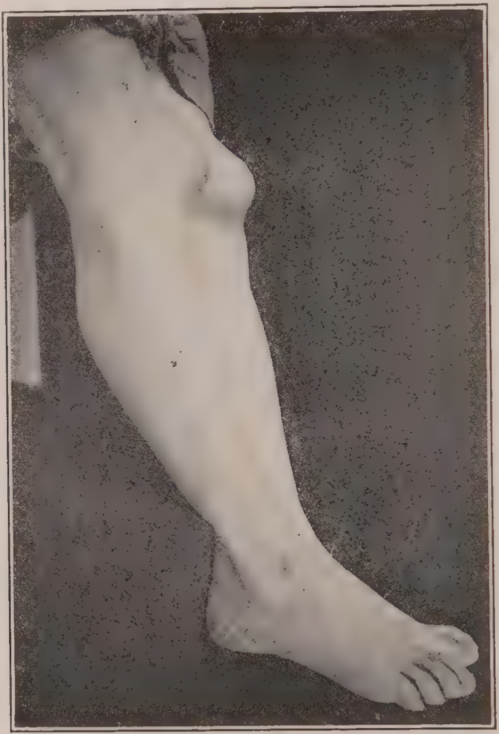

Fig. 243.—Prepatellar Bursitis ("Housemaid's Knee").

5. *The Lower Leg*

1. *Varicose veins*, with their results (eczema and ulcer), are the commonest lesions of the lower leg. The soft, twisted, purplish eminences are easily recognized. *Hardness* in such a vein usually means thrombosis. It should be remembered that pregnancy and pelvic tumors may produce varicose veins in the legs.

2. *Chronic ulcers of the lower leg*, especially those in front, are usually due to varicose veins and the resulting malnutrition of the tissues. They leave a *brown scar* after healing. Syphilitic ulcers

usually leave a white scar; they may occur in the same situation, but are more common above the knee or on the calf.

3. *Syphilitic periostitis* is common on the shaft of the tibia, and gives rise to *pain* (worse at night) with tenderness and some swelling. Later *bony nodes* are sometimes formed, similar to those already pictured on the frontal bone. In doubtful cases of syphilis in other parts of the body we may sometimes secure convincing evidence by radiography of the tibiæ. Periosteal thickening, not otherwise recognizable, may be thus brought to light and may help our diagnosis of a cardiac arthritis or hepatic lesion.

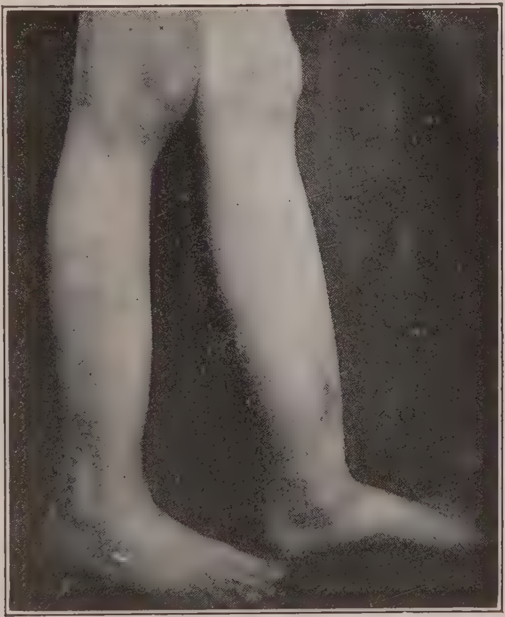

FIG. 244.—Syphilitic Periostitis ("Saber Shins").

4. *Osteomyelitis* (septic, tuberculous) often starts on the head of the tibia, with intense pain, tenderness, fever, and leucocytosis (if acute); "rheumatism" is often falsely diagnosed; there results a genereal septicæmia or a local sinus leading to dead bone.

5. *Sarcoma* not infrequently attacks the upper end of the tibia or fibula, producing lesions similar to those described in the femur. Secondary or metastatic tumors are often to be detected in the thigh bones and pelvic bones (see Figs. 246, 247, 248).

6. *Œdema of the legs*[1] is oftenest due to:

(*a*) Uncompensated heart lesions, primary or secondary from lung disease.

(*b*) Nephritis.

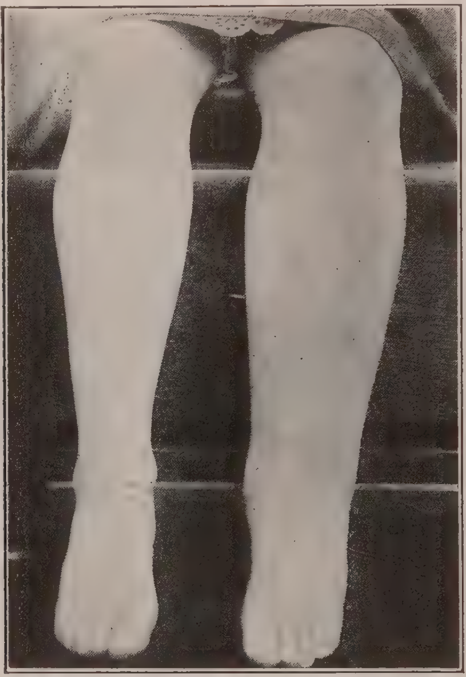

FIG. 245.—Angioneurotic Œdema of One Leg.

(*c*) Cirrhotic Liver.

(*d*) Anæmia.

(*e*) Neuritis (alcoholic, beri-beri, etc.).

(*f*) Varicose veins.

(*g*) Obesity, flat-foot, and other causes of deficient local circulation.

In some cases no cause can be found ("angioneurotic" œdema, "essential" and "hereditary" œdema). Diagnosis of the cause of œdema depends on the history and the examination of the rest of the body.

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[1] It is notable that œdema is usually greatest in the *front* of the leg and in the *back* of the thigh.

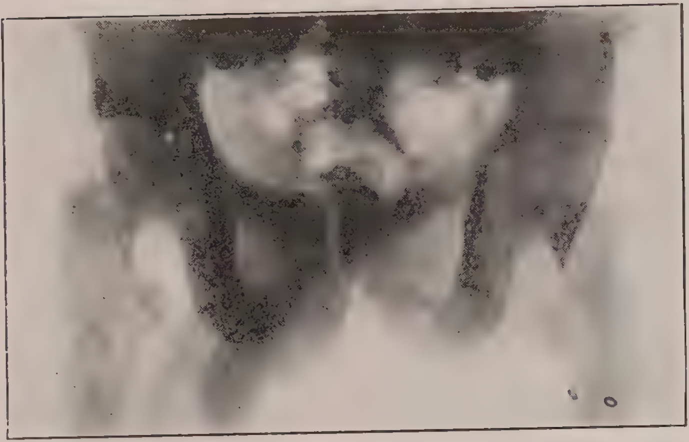

FIG. 246.—Metastatic bony tumors of the head of the right femur.

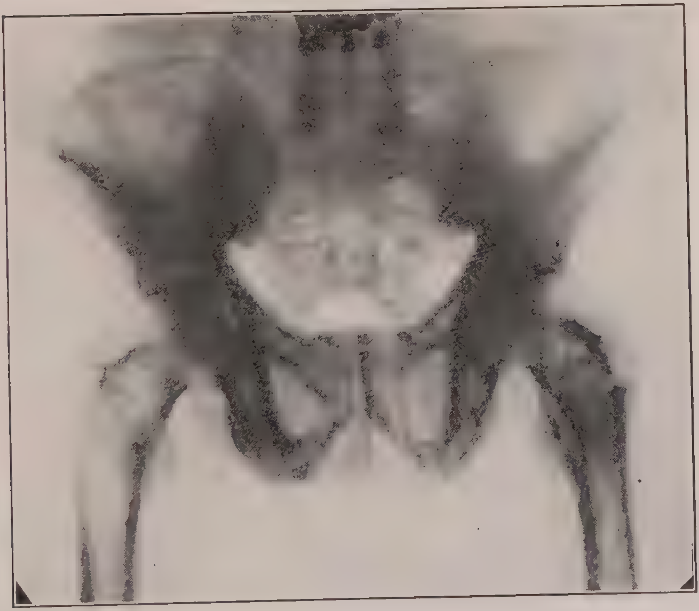

FIG. 247.—Metastatic bony tumors of the ischium pubic bone.

In one leg œdema may be due to *thrombosis* of a vein (see page 449), to pressure of *tumors in the pelvis* (pregnancy, etc.), to hemiplegia, elephantiasis (see Figs. 249 and 250) or to *inflammation.*

7. *Tenderness in the lower legs* frequently accompanies œdema from any cause. It may also be due to neuritis or trichiniasis, and, of course, to any local inflammation.

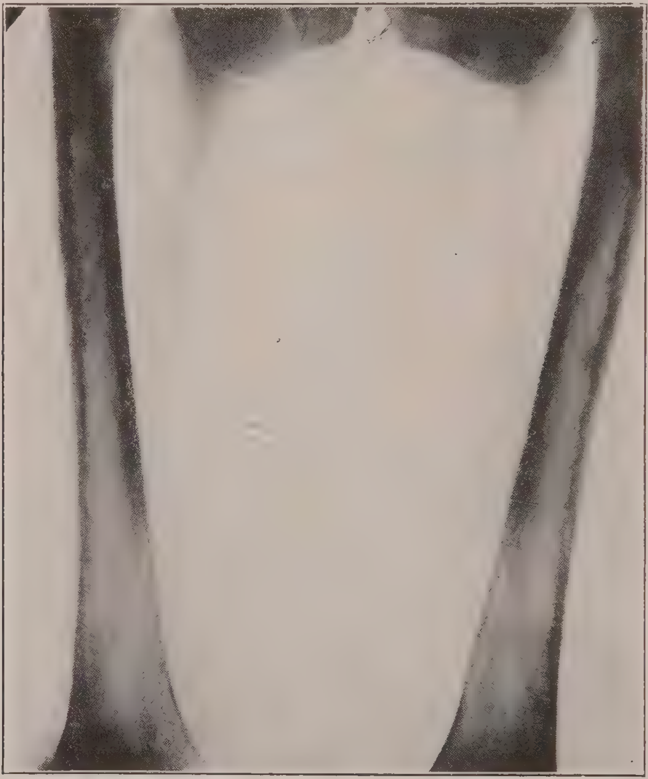

FIG. 248.—Metastatic bony tumors of upper one-half of both femora.

II. The Feet

1. The variaties of *club-foot* are: (*a*) *Equinus*, the heel drawn up. (*b*) *Varus*, the ankle bent outward. (*c*) *Valgus*, the ankle bent inward and the foot outward. (*d*) *Calcaneus*, the foot turned outward and upward.

The affection is usually congenital.

2. *Flat-foot* is a breaking down or weakening of the normal arch of the foot. There may or may not be changes in the sole-print. There may be pain and tenderness near the attachment of the ligaments and often higher up on the leg, but many cases are symptomless and should not be treated.

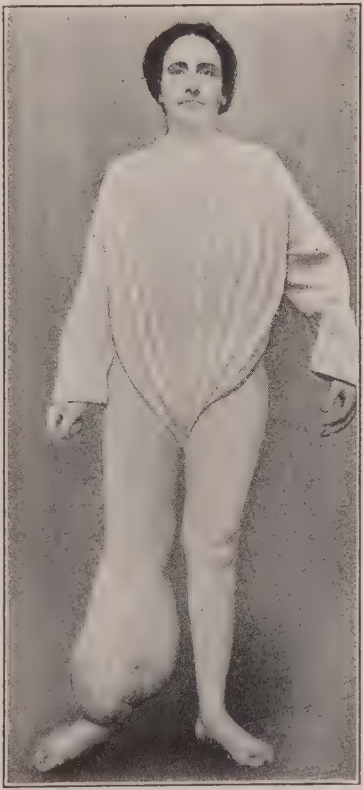

FIG. 249.—Sporadic elephantiasis. (Non-filarial.)

3. *Tenosynovitis of the Achilles tendon* often produces pain in the tendon, increased by use and sometimes associated with palpable creaking or crepitus over it.

4. *Enlarged (rachitic) epiphyses* are seen at the lower end of the tibia and fibula just above the ankle-joint in about forty per cent of rachitic cases. There may also be bending of the bones (see Fig. 252). The other signs of rickets in the child make diagnosis easy.

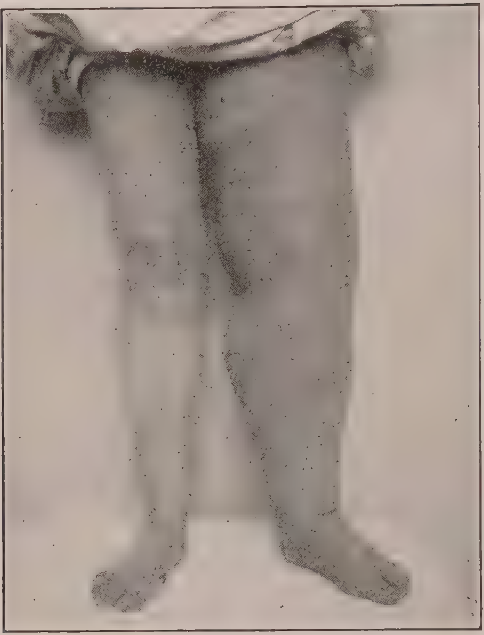

FIG. 250.—Elephantiasis.

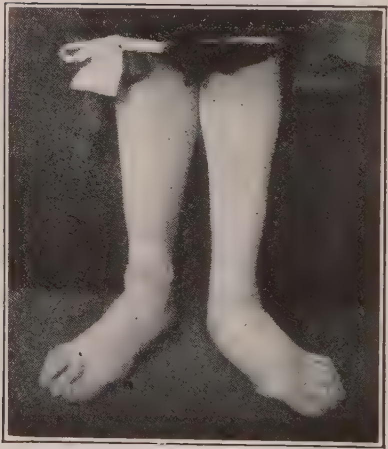

FIG. 251.—Flat-foot. (Bradford and Lovett.)

5. *Tuberculosis* is especially apt to attack the ankle bones in young persons. It is recognized by the usual evidences of joint tuberculosis (see below, page 483).

6. *Epithelioma* of the ankle has the characteristics of epithelioma elsewhere.

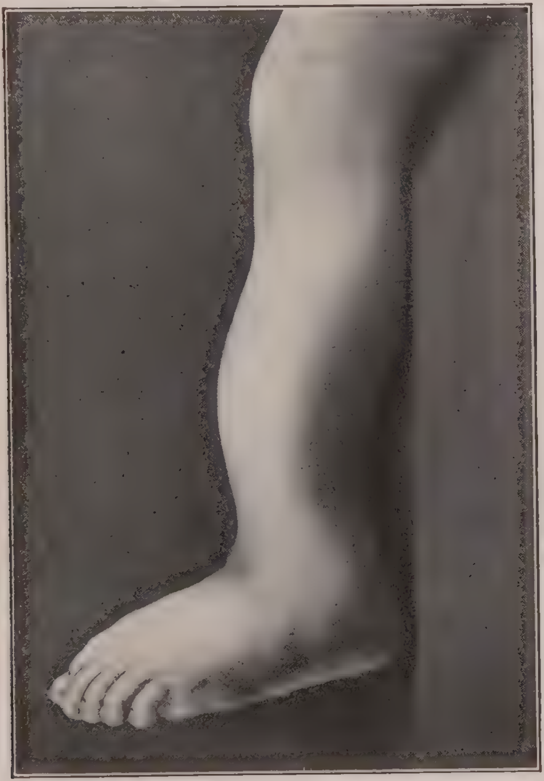

FIG. 252.—Rachitic deformity of leg bones.

7. *Erythromelalgia*, or red neuralgia of the extremities, is commonest in the feet. The toes (or fingers) are red, hot, tender, and painful. In Raynaud's disease the digits are cold and painless or anæsthetic. The attacks are aggravated by heat and not (like those of Raynaud's disease) by cold. Such attacks are probably akin to the condition of "*hot feet*" often seen in the arteriosclerosis of elderly people. The patient kicks off the bed clothes from his feet at night on account of

the burning sensations in them. Other evidence of insufficient arterial blood supply (*e.g.*, clubbing, intermittent claudication, cramps, gangrene) may coexist.

8. "Judaische Krankheit," an obliterating thrombosis of veins and arteries in the extremities, chiefly the feet, confined practically to the Jewish race, and usually resulting, after months of pain, in gangrene.

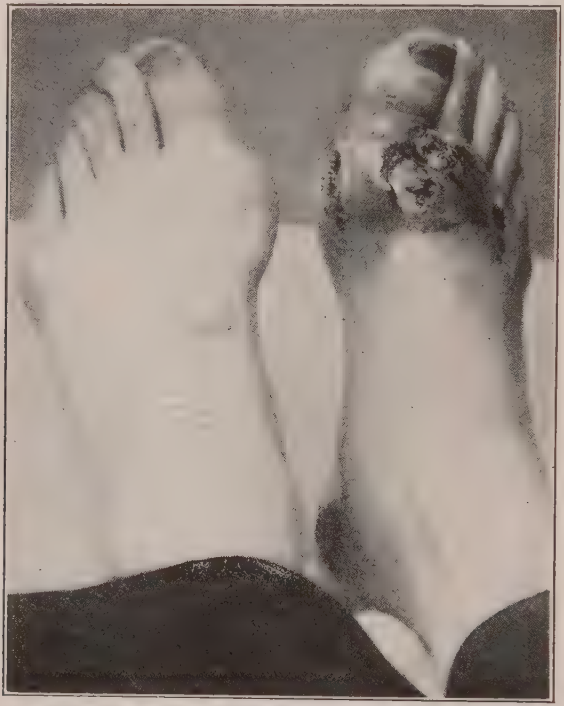

FIG. 253.—Arteriosclerotic gangrene.

1. *The Toes*

Many of the lesions already mentioned in the fingers are found also in the toes (*e.g.*, atrophic and hypertrophic arthritis, acromegaly, pulmonary osteoarthropathy, tuberculous or syphilitic dactylitis, tremors, spasms, and choreiform movements). Other lesions, such as ingrowing toe-nail, bunion, hallux valgus, policeman's heel, are too purely local to deserve description here. Excluding these we have left:

1. *Gout*, which is especially prone to attack the metatarso-phalangeal joint of the great toe, producing all the classical signs of inflammation and in chronic cases tophi (see page 494).

2. *Gangrene* is usually the result of arteriosclerosis (see Fig. 253) with or without diabetes mellitus, but may result (as in the fingers) from arterial spasm or local asphyxia (Raynaud's disease).

3. *Perforating Ulcer.*—In diabetes and sometimes in tabes a trophic or nutritional ulcer may develop in the toe or tarsus as a result of nerve influences similar to those which produce Charcot's joint or herpes zoster in the disease just mentioned. It is called "*perforating ulcer*" because of its stubborn progression despite a plan of treatment that checks ordinary infectious abscesses. Actual perforation is not often seen.

4. "*Tender toes*" after typhoid fever result from an infectious neuritis.

5. "*Morton's disease*" (metatarsalgia) means pain in the tarsus at a small spot near the distal end of one of the three outer toes, always associated with compression of the foot by tight boots and probably due to pinching of the external plantar nerves between the metatarsal bones. It is relieved by proper shoes.

CHAPTER XXVI

THE BLOOD

I. Examination of the Blood

The essentials of blood examination as a part of physical diagnosis are as follows:

I. *Hæmoglobin test* (Tallqvist) in all cases.

II. Study of a *stained blood film* in most cases.

III. *Total leucocyte count* (Thoma-Zeiss) in many cases.

IV. Count of *red corpuscles and Widal reaction* in a few cases.

I will now give a brief account of each of these methods and of the interpretation of the data obtained by them.

1. *Hæmoglobin*

(*a*) The Tallqvist scale consists of ten strips of red-tinted paper corresponding to the tint of a filter paper of standard quality when saturated with blood containing ten per cent, twenty per cent, thirty per cent, etc., hæmoglobin up to one hundred per cent. To perform the test we puncture the lobe of the ear with a glover's needle (*not* with sewing needle), saturate a strip of the filter paper which is bound up with the scale, in the blood of the patient to be examined, and compare the tint of this strip with the different standard tints in the scale. Let the blood dry until the gloss has disappeared. *Hold the blood spot beside not behind the scale.* Ignore the perforation in the latter. *Do not blot the blood spot*, and do not delay in making the comparison after the humid gloss has disappeared. Stand with the light behind you or at one side of you; use daylight always.

The test is not accurate within ten degrees, but a degree of accuracy greater than this is very rarely required for any purpose of diagnosis, prognosis, or treatment. In rare cases, when a more accurate reading is needed, we may use the instrument of Gowers as modified by Sahli.

(*b*) Sahli's instrument (see Fig. 254) would supplant all others but for the unfortunate fact that the standard solution is likely to become inaccurate in color. To use the instrument we first put a few drops of decinormal HCl solution into the empty tube (Fig. 254, *B*), so as to fill it to the mark 10; then suck up blood with the pipette (Fig. 254, *C*), until the mark 1 is reached. Wipe the point of the pipette and immediately blow out the blood into the solution at the bottom of the tube (*B*). Suck this mixture of blood and water back into the pipette and blow it out again twice to cleanse the pipette. Next add water from the dropper (*D*), a few drops at a time, until the tint of the mixture of the blood and water is the same as that of the standard solution, when both are looked at with transmitted light. After each addition of water close the end of the tube with the thumb and invert it twice, then scrape the thumb on the edge of the tube so as to rub off any moisture deposited there during the process of inversion. As the tint of the mixture of blood and water approaches that of the standard solution, add the water two drops at a time, and close the eyes for a few seconds between each two attempts at reading. When the colors in the two tubes seem to be identical, read off the figure corresponding with the meniscus of the column of fluid in the tube. The resulting figure represents the percentage of hæmoglobin.

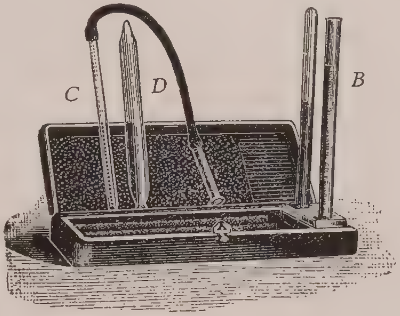

FIG. 254.—Sahli's Hæmoglobinometer. *B*, Diluting tube; *C*, pipette; *D*, dropper.

(*c*) *The Color Index.*—The data to be obtained by these instruments stand for the amount of the coloring matter in a given unit of blood when compared with the amount in a similar unit of normal blood. When the hæmoglobin percentage is low, anæmia is always present, and the degree of anæmia is measured by the amount of reduction in the hæmoglobin per cent. But the percentage of hæmoglobin is not a measure of the number of corpuscles present in a given unit of blood, for if the corpuscles are large and contain each of them a relatively large amount of hæmoglobin, they may be considerably diminished in number and yet furnish a normal bulk of hæmoglobin, as tested by either of the instruments described. Thus in pernicious anæmia the corpuscles are often so large that they contain nearly one-third as much again as a normal corpuscle, so that even though their number is considerably diminished they may carry a normal amount of hæmoglobin. This condition is known as a "*high color index.*" On the other hand, the number of red corpuscles may be normal, yet each corpuscle so deficient in hæmoglobin that the hæmoglobin in a given quantity of blood is as low as forty or fifty per cent. This state of things is often found in chlorosis or in any form of secondary anæmia (see below, page 472). When the diminution in the number of red corpuscles is greater than the diminution of hæmoglobin, we say that the color index is high, meaning that each corpuscle carries more hæmoglobin than normal. Thus if we have a red count of two millions and a half of red cells, and each cell contained the normal amount of hæmoglobin, the hæmoglobin percentage would be fifty, representing a reduction in hæmoglobin proportional to the reduction in the red cells; but if with the same count we had a hæmoglobin percentage of seventy-five, this would mean that each corpuscle contained half as much again as compared with the hæmoglobin in normal red cells. Here we should say that the color index is 1.5 Five million red cells and one hundred per cent. of hæmoglobin give a color index of 1. Four million red cells with forty per cent. hæmoglobin, represents a color index of 0.5; three million red cells with 40 per cent. hæmoglobin, represents a color index of 0.58.

The diagnostic significance of the color index is briefly this: *Any diminution in hæmoglobin means anæmia, but a diminution in hæmoglobin with a high color index suggests, though it does not prove, pernicious anæmia, while a low color index points to chlorosis or secondary anæmia of any type.* Normal color index, despite anæmia, is most often found immediately after hemorrhage.

Achromia shown in the stained film is the best obtainable evidence of low color index. When the film contradicts the hemoglobin measurement always believe the film.

2. *Study of the Stained Blood Film*

To recognize the *presence* and the *degree* of anæmia one needs only the *hæmoglobin test*, but to determine the *kind* of anæmia, to study the leucocytes, or to search for parasites we need the stained blood film. Two processes are now to be described:

1. Preparing the film.
2. Staining.

1. *Blood films* may be spread on slides or on cover glasses. The first method is the easier; the second gives better preparations. To prepare blood films on slides, dip two slides in water and rub them clean with a towel or handkerchief. Puncture the lobe of the ear (*not the finger*) with a bayonet pointed Glover's needle or surgical

needle. Put a drop of blood near one end of one slide, put the other slide against the drop, and rest it evenly upon the first, as shown in Fig. 255, so that the drop will spread laterally across the face of the "spreader." Next draw the upper slide along horizontally, so as to spread the drop over the whole surface of the lower slide. The process may then be repeated, reversing the slides and using as a "spreader" the one on which the film has already been prepared. Both slides are then allowed to dry in the air *without touching each other*. This method is so simple that one can usually succeed with it at the first attempt, but the corpuscles are not spread quite so evenly as in cover-glass preparations and it is somewhat more difficult to get a perfect stain.

The *cover-glass method* requires a much greater degree of cleanliness and manual dexterity than the slide method. Cover glasses must be washed in water and then *thoroughly* polished with a *silk* (not cotton or linen) handkerchief. The success of the whole process depends upon the thoroughness of the polishing. Every part of the glass must be thoroughly gone over, taking care not to omit the corners. This is rather tedious and often drives us to use slides, which can be much more quickly prepared. With cover glasses we must remove not only all dirt and grease, but also every speck of dust or lint which may settle upon them. The use of silk as a polisher reduces this difficulty to a minimum.

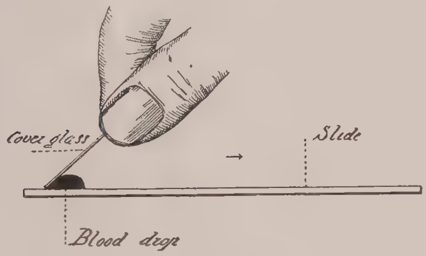

FIG. 255.—Method of Spreading Blood Films.

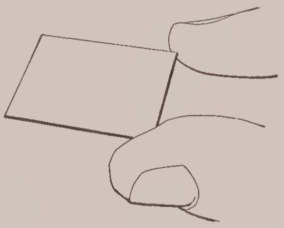

FIG. 256.—Proper Method of Holding a Cover Glass.

Having prepared the cover glasses in this way, the next point is to keep them both clean and dry during the process of spreading the blood. We must always hold them as in Fig. 256, and never touch any part of their surfaces with the fingers. Any one whose fingers tend to get moist must handle the cover glasses with forceps, but most of us will always use our fingers, despite the warnings of our Teutonic brethern. Holding a cover glass as in Fig. 256, touch the centre of it with the tip of a drop of blood as it issues from a puncture, taking care not to touch the skin of the ear itself; then drop this cover glass (blood side downward) upon a second cover glass in such a position that their corners do not match. If the covers are quite clean and free from dust, the blood drop will at once spread so as to cover the whole surface of the glasses. The instant it stops spreading, take hold of the upper cover glass by one corner and slide it rapidly off without lifting it or tilting it at all. This needs some practice, and some men never learn it; hence the use of slides.

Films so prepared will keep for a long time without deteriorating, especially if the air is excluded.

2. *Staining.*—The introduction of the Romanowsky method of staining (Nocht's, Ziemann's, Jenner's, Leishman's, Wright's) enables us to dispense with all other blood stains and greatly shortens the time of the process. Wright's stain is identical with Leishman's except in the method of preparation, which Wright has considerably simplified, and as either of these mixtures can be obtained ready made of any of the larger dealers in physicians' supplies, I shall not describe the method of making it. Reliable stains can always be obtained from the Massachusetts General Hospital in Boston. An ounce bottle will stain hundreds of specimens.

To stain a cover-glass film, grasp it with Cornets's forceps, rest the forceps on the sink so that the film side is upward and is approximately horizontal. Draw a little of Wright's or Leishman's stain into a clean medicine-dropper and squeeze out upon the film enough to flood its surface.

(*a*) Allow the stain to *act for one minute;* during this time the methylic alcohol contained in it fixes the film upon the cover glass.

(*b*) Next add distilled water from a clean medicine-dropper until a greenish metallic lustre appears like a scum upon the surface of the stain. Usually about six or eight drops of water are needed if we are using a seven-eighths-inch cover glass. The stain, so diluted with water, should remain upon the cover glass about *two minutes*. The exact time does not matter.

(*c*) Next wash off the stain with water cautiously and let the film remain in clean water for about a minute more or until it takes on a light pink color. Dry gently with blotting paper and mount in Canada balsam.

This whole process can be completed inside of five minutes, and I know of no other staining method at once so rapid, so reliable, and so widely applicable. It brings out all the minutiæ of the red corpuscles, leucocytes, and blood parasites and for clinical work no other stain is needed.

Appearance of Films so Stained.—1. The *normal red corpuscles* appear as round discs with pale centres. Their color depends upon the length of time that we continue the washing with clear water after the staining mixture has been poured off, and varies from brown through pink to golden yellow.

(*a*) *Poikilocytosis* means the appearance in the blood of red cells variously deformed, sausage shaped, battledore shaped, oblong, pear shaped, etc. It is always associated with *abnormalities in the size* of the corpuscles, so that dwarf forms and giant forms appear.

(*b*) *Polychromasia* (or *polychromatophilia*) refers to abnormal staining reactions in the red corpuscles, whereby isolated individuals take on a brownish or purplish tint, sharply contrasted with the pink or yellow of the corpuscles around. If this brownish or purplish tint occurs in all the corpuscles, it has no pathological significance, but merely means that the staining has been incorrectly performed.

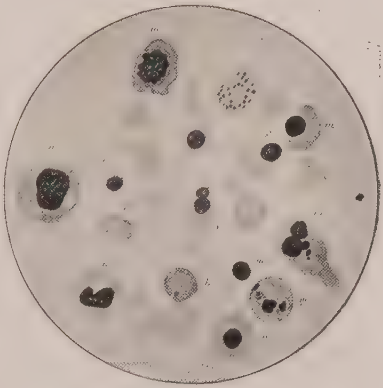

Fig. 257.—Nucleated Red Cells. *m, m*, Megaloblasts; *n*, normoblast; *s*, stippled cell.

(*c*) "*Stippling*" refers to fine, dark-blue dots scattered over the pink surface of a red corpuscle, as if a charge of fine shot had been fired into it.

All the abnormalities just described are to be found in any of the types of severe anæmia, whether primary or secondary, but *stippling* may also be found *without anæmia in* some cases of *lead poisoning*, and is therefore useful as a confirmatory sign in cases of this disease.

Nucleated red corpuscles are divided into two main varieties: (1) *normoblasts*, which are of the size of normal corpuscles; and (2) *megaloblasts*, which are larger than normal corpuscles (see Fig. 257). The nucleus of the normoblast is generally small and deeply stained, navy blue. In the megaloblast the nucleus may have the same characteristics or may be much larger and paler, with a distinct intranuclear network. The protoplasm of both varieties is often discolored, murky, gray, or even blue, and sometimes stippled, so that by beginners the cell may be mistaken for a leucocyte. The mistake may be avoided, however, after some experience. In the protoplasm of nucleated cells there are often concentric rings like the layers in an oyster shell, and their outline is usually more irregular than that of any leucocyte. Further points of differentiation must be learned by practice.

2. *Leucocytes.*—In normal blood four main varieties may be distinguished:

(*a*) Polynuclears or polymorphonuclear neutrophiles.
(*b*) Lymphocytes (including endothelial cells).
(*c*) Eosinophiles.
(*d*) Mast cells.

(*a*) *Polynuclears.*—The deeply stained, markedly contorted nucleus assumes a great variety of shapes in different cells, and is surrounded by a pinkish protoplasm studded with spots or granules just large enough to be distinguished under the oil immersion and slightly deeper in tint than the protoplasm around them. These cells make up about two-thirds (*fifty to seventy per cent*) of all the leucocytes present in the blood (see Fig. 258, *a*).

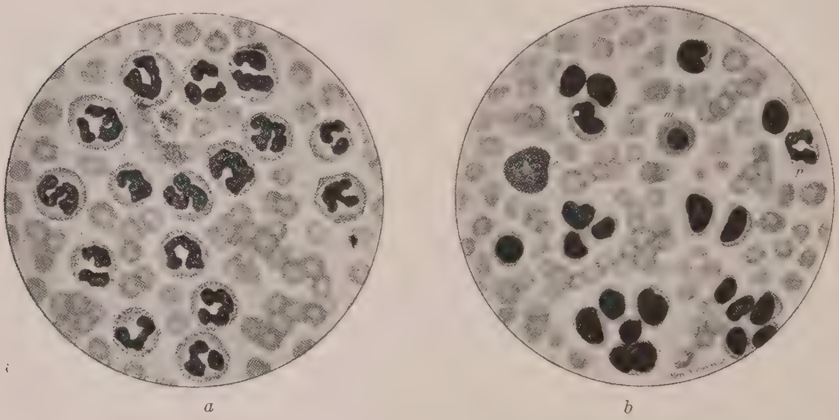

FIG. 258.—*a*, Leucocytosis (40.000); sixteen polynuclears in a field. *b*, Lymphatic leukæmia. *p*, Polynuclear; *m*, megaloblast; *e*, eosinophile. Twenty-one lymphocytes in this field.

(*b*) *Lymphocytes.*—The smallest variety is about the size of a red cell, and consists of a round nucleus stained deep blue and surrounded by a very narrow rim of pale, bluish-green protoplasm. In the larger forms (endothelial cells) the nucleus occupies less space, is often less deeply stained, and may be indented. The latter variety is sometimes burdened with the useless name of "transitional cell," a term which in my opinion should be given up, since all lymphocytes are transitional. In the protoplasm of the larger varieties of lymphocyte one often sees a sprinkling of fine pink granules. From thirty to fifty per cent (or about one-third) of all leucocytes belong to the lymphocyte group—classing all sizes together (see Fig. 258, *b*).

(*c*) *Eosinophiles.*—The nucleus is irregularly contorted and attracts very little notice, owing to the very brilliant pink color and relatively large size of the granules in which it is immersed. The outline of the cell is more irregular than that of any other leucocyte, and its granules often become broken away and scattered in the technique of spreading the blood. The *eosinophiles* make up approximately *one per cent* of the leucocytes of normal blood.

(*d*) *Mast Cells.*—The shape of the nucleus can rarely be made out, and the main characteristic of the cell is the presence of large dark granules, stained bluish black or plum color, and arranged most thickly about the margin of the cell. Mast cells are very scanty in normal blood and make up not more than *one-half of one per cent* of the leucocytes.

Other varieties of leucocytes which appear in the blood only in disease will be mentioned later.

3. *Blood Plates.*—In the normal blood film, stained as directed above, one finds, beside the red corpuscles and the different varieties of leucocytes, a varying number of bodies, usually about one-third the diameter of a red corpuscle, irregularly oval in shape, staining dark red or blue and tending to cohere in bunches. Occasionally larger forms occur, and in these a vague network and some hints of a nucleus may be traced.

These bodies which are probably derived from one or more species of leucocytes have at present no considerable importance in medicine, although they not infrequently lead to mistakes, because, when lying on top of a red corpuscle, they bear a slight resemblance to a malarial parasite. They are usually increased in secondary anæmia and diminished in pernicious anæmia.

(*a*) *The Differential Count of Leucocytes*

A film stained as above directed is moved past the objective of the microscope either with a mechanical stage or with the fingers, and every leucocyte seen is classified under one of the heads just described until from 200–400 leucocytes have been thus differentiated. The percentages are then reckoned out.

The points most often looked for are:

1. Increase in the per cent of polynuclears.
2. Increase of eosinophiles.
3. Increase of lymphocytes.
4. Presence of myelocytes and other abnormal forms (see below).
5. Changes in the red cells noted simultaneously.

3. *Counting the White Corpuscles*

The instrument used all over the world at the present day is the pipette of Thoma-Zeiss, in which the blood is diluted either ten or twenty times. The diluting solution is one-half of one per cent glacial acetic acid in water. This diluting solution often accumulates spores and becomes cloudy. As soon as this happens a fresh bottle should be prepared. After a rather deep puncture blood is sucked up to the point marked .5 on the pipette, which is then immersed in the diluting solution and suction exerted until the mixture is drawn up to the point marked 11. This gives a dilution of one to twenty. By drawing blood up to the point marked 1, instead of to the point marked .5, we obtain a dilution of one to ten. After this the ends of the pipette can be closed with a rubber band, and the blood, so shut in, can be kept or transported without loss or change.

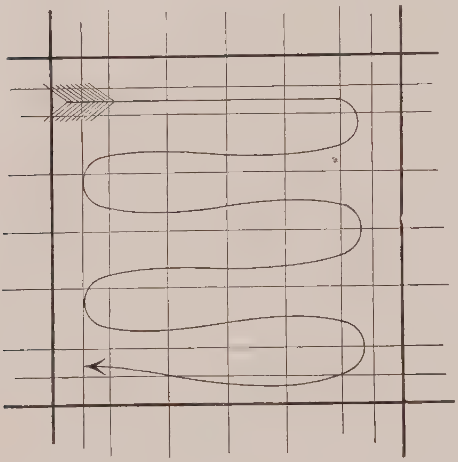

FIG. 259.—Indicating an Order in which the Squares may be Counted.

When we are ready to make the count, the rubber band is removed and the pipette rolled in the fingers rapidly back and forth for about one minute, to mix up the contents of the bulb thoroughly and evenly. Next blow out three drops, in order to get rid of the pure diluting solution which is in the shank of the pipette. Then put upon the circular disc of the counting chamber a drop of the mixture from the bulb of the pipette. This drop must be of such a size that when the cover glass (see Fig. 260, *B*) is let down upon it[1] the drop will cover at least nine-tenths of the circular disc and not spill into the moat around it. The size of this drop can only be learned by practice. After about five minutes the leucocytes will have settled upon the ruled space which occupies the centre of the floor of the counting chamber, and the count can then be begun, using preferably a No. 5 objective of Leitz or a DD of Zeiss. The whole ruled space should be counted, and after a little practice this takes not more than five minutes. I usually begin my count in the left upper corner of the ruled space and proceed in the direction indicated by the serpentine arrow in Fig. 259. In normal blood one finds from thirty to fifty leucocytes in the whole ruled space. The number of leucocytes per cubic millimetre is obtained by multiplying this figure by 200. Thus if the number of leucocytes counted is 35, the number in a cubic millimetre of blood is 35 × 200 = 7,000. If great accuracy is needed, a second count with a fresh drop should be made and the average of the two taken; but in ordinary clinical work this does not seem to me necessary, for the amount of error, although considerable, is not such as to affect our diagnostic inferences.

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[1] To avoid air bubbles lower the cover glass with aid of a needle as in mounting microscopic specimens. This must be done as quickly as possible after the drop has been adjusted on the counting disc.

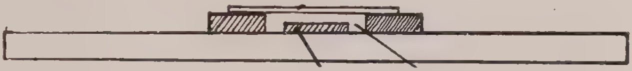

FIG. 260.—Thoma-Zeiss Counting Slide. *A*, Ruled disc; *B*, cover-glass; *C*, moat.

4. *Counting the Red Corpuscles*

Perhaps once in every twenty-five or fifty cases that one sees it is well to know the number of red corpuscles. They can then be counted with the Thoma-Zeiss pipette which is made for the purpose, and so arranged that the blood may be diluted one to two hundred. The technique is exactly that described in the last section, except that we need less blood and use a different diluting solution. I am accustomed to use a mixture suggested by Gowers, made up as follows:

Sodium sulphate	gr. cxii.
Dilute acetic acid	ʒi.
Water	℥iv.

Blood is sucked up to the mark 0.5 and then Gowers' solution to the mark 101. After the drop has been adjusted in the counting chamber and the corpuscles have settled upon the ruled space, we usually count a field of twenty-five small squares at each of the four corners of the whole ruled space. The figure so obtained is multiplied by 8,000. The result is the number of corpuscles per cubic millimetre.

II. Interpretation of the Results so Obtained

1. *Secondary Anæmia*

The hæmoglobin is usually reduced more than the count of red corpuscles giving a *low color index.* In mild cases the hæmoglobin may fall as low as forty per cent before the red corpuscles show any considerable diminution. In severe cases the red cells fall to 3,000,000, 2,000,000, and occasionally even to 1,000,000 or below it; but the hæmoglobin usually suffers even more severely.

The leucocytes may be normal, increased, or diminished, depending on the cause of the anæmia. Thus in anæmia due to chronic suppurative hip-disease the leucocytes are often increased to 20,000 or 30,000, while in malarial anæmia the leucocytes are often subnormal. There are no characteristic changes in the differential count, which varies with the underlying disease.

The changes seen in the red cells in the stained blood film are briefly: *Achromia;* sometimes polychromasia, stippling, poikilocytosis, and the presence of nuclei either in normal-sized corpuscles (normoblasts) or in giant corpuscles (megaloblasts). *Achromia or abnormally great pallor of the centres of the cells is the most important point in the recognition of secondary anæmia and the exclusion of pernicious anæmia.* An occasional normoblast or rarely a megaloblast can be found.

The commonest causes for secondary or symptomatic anæmia in adults are as follows:

(*a*) Hemorrhage—gastric, hemorrhoidal, traumatic, puerperal, etc.
(*b*) Malaria, more rarely sepsis or other infections.
(*c*) Malignant disease.
(*d*) Chronic suppurations.
(*e*) Chronic nephritis.
(*f*) Cirrhosis of the liver.
(*g*) Poisons, especially lead.
(*h*) Chronic dysentery.
(*i*) Intestinal parasites.

It is important to remember that insufficient food or even starvation does not produce anæmia, and so far as we know no form of bad hygiene has any notable effect upon the blood. Persons may grow very pale under bad hygienic conditions, but their blood is usually not affected unless one of the diseased conditions mentioned above is present.

2. *Chlorosis*

The blood is practically identical with that just described, though the color index is sometimes lower, poikilocytosis less marked, and nucleated red cells fewer. The pallor of the centres of the cells ("achromia") is often very marked. The leucocytes are generally normal and the differential count practically so, although the percentage of polynuclear cells is often low with a corresponding relative increase of lymphocytes.

3. *Pernicious Anæmia*

The number of red cells is usually below 2,000,000 when the case is first seen. The color index is high and the leucocyte count subnormal. In the stained specimen *the essential point is the prevalence of big non-achromic red cells;* there are also very marked deformities and abnormal staining reactions in the red cells. A few of the large reds contain nuclei ("megaloblasts"), and a number of normal-sized cells also contain nuclei ("normoblasts").

The polynuclears are absolutely diminished, with a corresponding relative increase in the lymphocytes.

In the remissions which form so important a feature of the course of pernicious anæmia, the blood is generally transformed until it contains approximately 100 per cent. of hæmoglobin, although the red cells seldom get above 4,000,000. The abnormally large and deeply stained red cells still prevail in most cases and make diagnosis possible though difficult.

(*a*) *Interpretation of the Results of the Leucocyte Count and Differential Count*

By combining the facts obtained by the total white count and the differential count, we can estimate the number of each variety of leucocyte contained in a cubic millimetre of blood. Thus with 10,000 white corpuscles, 70 per cent. of which are polynuclear (as seen in the stained film), we have 7,000 polynuclear cells per cubic millimetre, which may be considered the upper normal limit. Any number greater than this should be considered as a *leucocytosis*. In a similar way we can say that any number greater than 3,500 is above the normal limit for lymphocytes and constitutes a *lymphocytosis*, while *eosinophilia* is present whenever the number of eosinophiles is more than 400 per cubic millimetre. It is much better to use these absolute numbers than to rely upon percentages. If we say, for example, that 3 per cent of eosinophiles is within normal limits, we shall make an error now and then in cases of myelogenous leukæmia, in which, with a total count of 500,000 leucocytes, 3 per cent of eosinophiles would amount to a total of 15,000 per cubic millimetre, or nearly thirty times the normal number. Errors are also common in the opposite direction. For example, in typhoid, with a total leucocyte count of 3,000, the lymphocytes may reach 60 per cent and yet be well within the normal limits, for 60 per cent of 3,000 is only 1,800. In this case the apparent lymphocytosis is due to an *absolute decrease* in polynuclear cells.

For the reasons here given it seems to me best to use the following definitions:

1. *Leucocytosis* is an increase in the polynuclear cells beyond the normal—7,000.
2. *Lymphocytosis* is an increase of lymphocytes beyond the normal upper limit—3,500.
3. *Eosinophilia* is an increase of eosinophiles beyond the normal upper limit—500 per cubic millimetre.

4. *Occurrence of Leucocytosis*

Leucocytosis, like fever, occurs in a great variety of conditions, of which the following are the most important:

1. In *infectious diseases—except* typhoid, typhus, malaria, uncomplicated tuberculosis, measles, smallpox (prior to the pustular stage), mumps, German measles, and influenza (most cases).
2. In a variety of *toxæmic conditions*, such as uræmia, hepatic toxæmia, diabetic coma, rickets, and poisoning by illuminating gas.
3. In a minority of cases of *malignant disease*, especially sarcoma.
4. After *violent muscular exertion*, including parturition, after cold baths or massage, hæmorrhage and apoplectic seizures.

There is in all probability no constant leucocytosis in pregnancy or during digestion.

Leucocytosis is most often of value in the differential diagnosis between typhoid fever or malaria on the one hand, and pyogenic infections (meningitis, appendicitis, sepsis, pneumonia) on the other. A leucocyte-chart is often of value in judging whether a local suppurative process, such as appendicitis, is advancing or receding, or whether pus-pocketing has taken place. By a leucocyte-chart is meant series of leucocyte counts at short intervals—twelve, twenty-four, or forty-eight hours. *When taken in connection with the other clinical data*, a leucocyte chart is often of the greatest value, especially in following the *course* of any disease; to a less extent in diagnosis. Sondern (*Med. Record*, N. Y., March 25, 1905) considers that the higher the per cent of polynuclears, the severer the infection, while the body's resistance is mirrored in the height of the *total* leucocyte count. By noting both these facts, therefore, we have a prognostic guide of some importance. Most subsequent observations have tended to verify Sondern's theory. In internal medicine leucocyte counts are especially useful in *febrile conditions*, in the great majority of which they assist the diagnosis.

Certain exceptions to the rules above given must be remembered:

1. *Quiescent*, thickly encapsulated collections of *pus*, in which the bacteria have died or lost their virulence, usually *produce no leucocytosis*. In this group come some of the abscesses of the liver or about the kidney, and a few cases of appendicitis.

2. *The most virulent* and overwhelming *infections* are apt *not* to be *accompanied by leucocytosis*. Thus, for example, the most virulent cases of pneumonia, diphtheria, or general peritonitis often run their course without leucocytosis.

5. *Lymphocytosis*

Only in three diseases does well-marked lymphocytosis often occur: 1. Lymphatic leukæmia. 2. Whooping-cough and its complications (many cases). 3. Acute sepsis especially tonsillitis with or without glandular enlargement may produce a lymphocytosis which, but for the etiological factor, would be alarmingly like lymphoid leukæmia.

Occasionally lymphocytosis occurs in rickets, hereditary syphilis, and anything that produces debility in children. Lymphocytosis is of value chiefly in the differentiation of lymphatic leukæmia from other causes of chronic glandular enlargement.

6. *Eosinophilia*

The eosinophiles are increased chiefly in:

1. Bronchial asthma.
2. Chronic skin diseases.
3. Diseases due to animal parasites (trichiniasis, uncinariasis, filariasis, hydatid disease, Bilharzia disease, trypanosomiasis, and with most of the intestinal worms).
4. Myelogenous leukæmia.

There seems to be also some vague connection between eosinophilia and disease of the female genital tract (except cancer and fibromyoma of the uterus).

Leukæmia

Two forms are distinguished, though the distinction is chiefly a clinical one: (*a*) Myeloid and (*b*) lymphoid.

(*a*) *Myeloid Leukæmia*

The leucocytes are usually about 250,000 per cubic millimetre when the case is first seen, but often run much higher, and sometimes lower. There is no anæmia in the earliest stages; later moderate secondary anæmia develops.

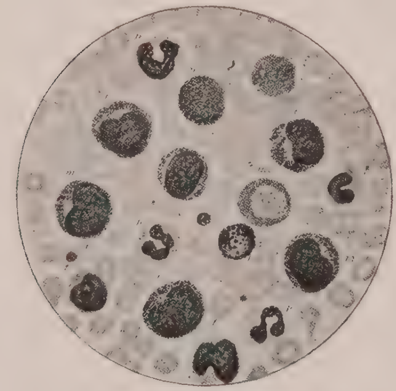

FIG. 261.—Myelogenous Leukæmia. *m*, Myelocytes; *p*, polynuclear; *b*, mast cell; *n*, normoblast.

The differential count shows an extraordinary *variety of types*, including many not seen in normal blood (see Fig. 261). The *majority* of the leucocytes are *polynuclears*, but many of these are atypical in size or in the shape of their nucleus. From 20 to 40 per cent of the leucocytes are *myelocytes* (or mononuclear neutrophiles), the "infantile" form of the polynuclear cell. *Lymphocytes* are absolutely normal or increased, but their percentage is low, on account of the greater increase of the other forms. *Eosinophiles* are absolutely much increased, though the percentage is not much above normal. *Mast cells* are more numerous than in any other disease (1 to 12 per cent, out of an enormous total increase). *Normoblasts* are usually very numerous; megaloblasts scanty.

Under the influence of intercurrent infections or after *x*-ray treatment the blood may return to normal.

(*b*) *Lymphoid Leukæmia*

The total increase of leucocytes is usually much less than in the other type of leukæmia—40,000 or 80,000—or less in average cases. The differential count shows an overwhelming proportion of lymph-

FIG. 1.—Young Tertian Parasites. (Stained with Wright's modification of Leishman's stain.)

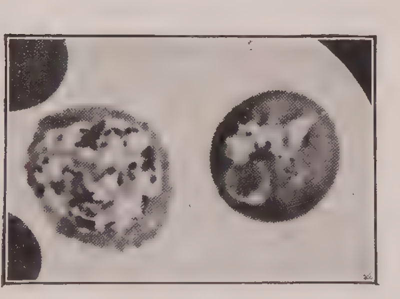

FIG. 2.—Mature Tertian Parasites. (Eosin and methylene blue.)

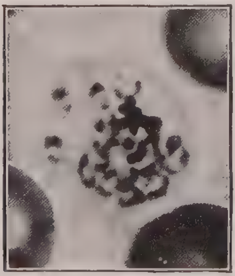

FIG. 3.—Segmenting Tertian Parasites. (Eosin and methylene blue.)

ocytes—90 to 99.9 per cent as a rule. In the acute forms of the disease the large lymphocytes predominate; in chronic cases the small forms.

Tonsillar infections and whooping-cough must be excluded before diagnosing lymphoid leukæmia from the blood film.

III. The Widal Reaction

(*a*) *Technique.* Among the numerous agglutinative reactions between the serum of a given disease and the micro-organism producing that disease, only one has yet attained wide use in clinical medicine, viz., the so-called Widal reaction in typhoid fever.

There are many ways of performing this reaction, but in my opinion the following is the best:

Measure out in two small test tubes ten drops and fifty drops respectively of a highly motile twelve- to twenty-four-hour bouillon culture of typhoid bacilli, in which the bacilli have no tendency to adhere spontaneously to each other. Carry these tubes and a microscope to the bedside, puncture the patient's ear as usual, and draw a little blood into a medicine-dropper of the same size as that used in measuring out the typhoid culture. Expel one drop of blood into each of the tubes containing typhoid culture, and examine a drop of each mixture between a slide and cover glass with a high-power dry lens. If within fifteen minutes clumping has taken place in the 1:10 mixture, or if within one hour clumping has taken place in the 1:50 mixture, the reaction may be considered positive. By clumping I mean an agglutination of the bacilli into large groups and the complete or nearly complete cessation of motility.

If it is inconvenient to carry the culture and the microscope to the bedside, ten or twenty drops of blood may be milked out of the ear and collected in a test tube (a three-inch test tube of small calibre is best). After clotting has taken place, if the edges of the clot are separated from the glass with a needle or a wire, a few drops of serum will exude, and this serum can be mixed with the bouillon culture in the manner already described.

Less reliable, in my opinion, is the use of blood dried upon glass or glazed paper in large drops and subsequently dissolved in the culture itself.

(*b*) *Interpretation.* A positive reaction occurs at some period in the course of ninety-five per cent of all cases of typhoid fever, but the proportion of cases in which the reaction occurs early enough to be of diagnostic value varies greatly in different epidemics. In most epidemics about two-thirds of the cases show a positive Widal reaction by the time the patient is sick enough to consult a physician. In the early days of the fever the Widal is often used but it is just at this period that we so often find typhoid bacilli by blood culture. Thus the two tests supplement each other.

IV. The Wassermann Reaction

I shall attempt no description of the technique of this most valuable and important test, because its performance is so difficult and delicate that only one who is constantly doing it is reliable. A positive reaction reliably done is important but not conclusive evidence of syphilis. Negative reactions do not exclude syphilis. The reaction is of especial value in cases of aneurism, aortic regurgitation, visceral and cerebral syphilis, doubtful cutaneous and arthritic and osseous lesions. Also in tabes and dementia paralytica.

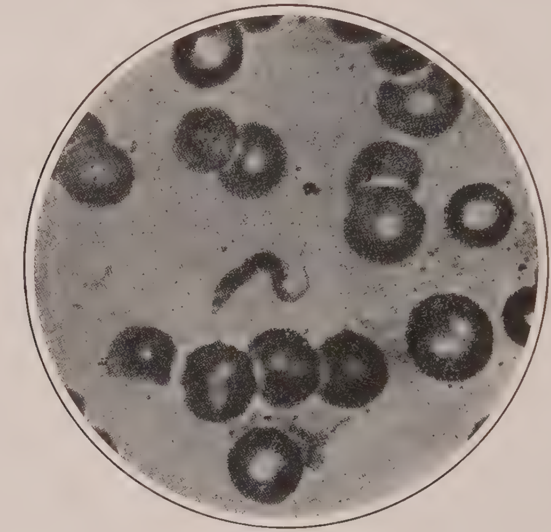

Fig. 262.—Trypanosoma in Human Blood. (By permission of Dr. J. Everett Dutton and the London *Lancet*.)

V. Blood Parasites

1. *The Malarial Parasite* (see Plates IV. and V.)

In films stained as above directed the malarial parasite appears blue against the pink background of the corpuscle. A crimson-stained dot should appear in some portion of the blue-stained organism;

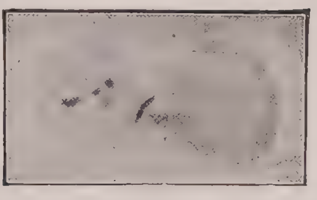

Fig. 1.—Two Young Æstivo-autumnal Parasites. (Wright's modification of Leishman's stain.)

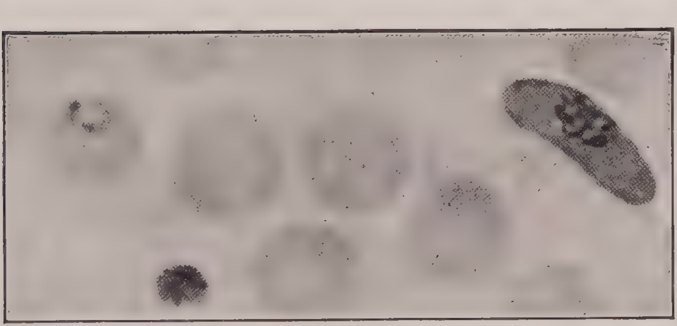

Fig. 2.—Æstivo-autumnal Parasites. Ring body at the left; crescent at the right. Stained like Fig. 1.

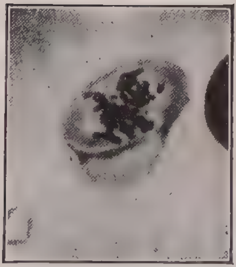

Fig. 3.—Ovoid in Æstivo-autumnal Malaria.

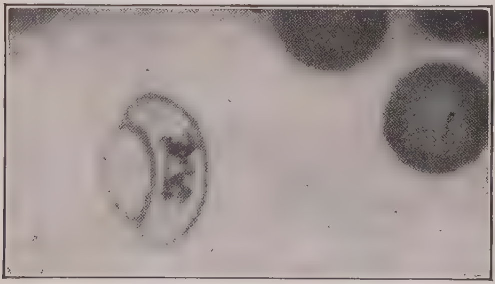

Fig. 4.—Crescent in Æstivo-autumnal Malaria.

the protoplasm of the red corpuscle around it is often studded with pink dots.

The stained specimen is preferable to the fresh blood in the search for malarial parasites, for the young, ring-shaped, or "hyaline" forms often escape notice altogether in fresh specimens.

Tertian organisms are distinguished from the æstivo-autumnal variety by the following tests:

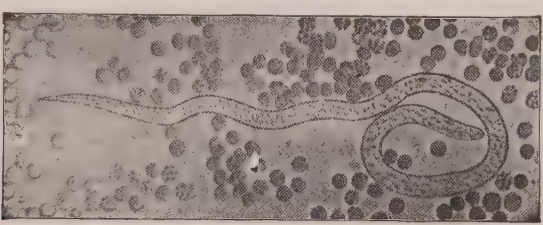

FIG. 263.—The Filaria Sanguinis Hominis. The head, curled up, is seen to the right of the cut, the tail to the left. Instantaneous photomicrograph. Four hundred diameters magnification.

(*a*) Tertian parasites make the corpuscle to which they are attached larger than its uninfected neighbors.

(*b*) Segmenting forms are rare in the peripheral blood of æstivo-autumnal fevers.

(*c*) "*Crescents*" (see Plate V.) never occur except in æstivo-autumnal fevers.

2. *The Trypanosomiasis*

In Central Africa (and presumably in other tropical countries) the blood or gland juice of many persons contains the organism shown in Fig. 262, which has long been known as a parasite of the blood of horses and of many of the lower animals. Human trypanosomiasis—a chronic debilitating malady—becomes "sleeping sickness" when the trypanosoma enters the cerebrospinal canal.

3. *Filariasis*

In the blood of many inhabitants of tropical countries there is found (with or without symptoms) the parasite shown in Fig. 263. The species most often found is present in the peripheral blood only at night; hence the blood should be examined after 8 P. M. A fresh drop is spread between side and cover and examined with a low-power lens (No. 5 objective Leitz).

CHAPTER XXVII

THE JOINTS

I. Examination of the joints

1. *Methods and Data*

I. By *inspection* and *palpation* we detect:

1. Pain, tenderness, and heat in, near, or at a distance from the joint.

2. Enlargement:

(*a*) Hard, probably bony.

(*b*) Boggy, probably infiltration or thickening of capsule and periarticular structures.

(*c*) Fluctuating, probably fluid in the joint.

3. Irregularities in contour:

(*a*) Osteophytes or "lipping" (attached to the bone).

(*b*) Gouty tophi (not attached to the bone).

(*c*) Constriction-line opposite the articulation.

(*d*) Protrusion of joint-pockets in large effusions, filling out of natural depressions.

4. Limitation of motion:

(*a*) Due to pain and effusion.

(*b*) Due to muscular spasm.

(*c*) Due to thickening or adhesions in the capsular and periarticular structures.

(*d*) Due to obstruction by bony outgrowths or gouty tophi.

(*e*) Due to ankylosis.

5. Excess of motion (subluxation).

6. Crepitus and creaking.

7. Free bodies in the joint.

8. Trophic lesions over or near a joint (cold, sweaty, mottled, cyanosed, white, or glossy skin, muscular atrophy).

9. Sinus formation, the sinus leading to necrosed bone, to gouty tophi, or absence in or near the joint.

10. Distortion and malposition, due to contractures in the muscles near the joint, to necrosis, to exudation, or to subluxation.

11. Telescoping of the joint with shortening (limb, toe, finger, or trunk).

II. By *radioscopy* we investigate:

1. Bony outgrowths, their shape, extent, and position.
2. Necroses and atrophies of bone, their extent and position.
3. The structure of the bones in and near the joints.
4. The presence of lesions in the articular cartilages.
5. Free joint bodies, their presence and position.

III. *Indirectly* we may gain valuable information about the joints by noting:

1. General constitutional symptoms, their presence or absence. These include fever, chills, leucocytosis, glandular enlargement, albuminuria, and emaciation.
2. Tuberculin reaction and Wassermann reaction, perhaps gonococcus fixation test,—their presence or absence.
3. Disease of other organs, their presence or absence, *i.e.*, syphilis, tuberculosis, tabes, and other chronic spinal-cord lesions, endocarditis, hæmophilia, various acute infections (gonorrhœa, influenza, scarlatina, septicæmia), and skin lesions (psoriasis, purpura, hives).
4. The course of the disease and the results of treatment.

2. *Technique of Joint Examination*

(*a*) *Enlargement* is generally unmistakable, but when there is much muscular atrophy between the joints the latter may seem enlarged by contrast, when in fact they are not.

(*b*) *Fluctuation* is obtained in most joints, as in any part of the body, by pressing a finger on each of two slightly separated spots in the suspected area, and endeavoring to transmit through the intervening space an impulse from one finger to the other. Fat or muscle will also transmit an impulse, but less perfectly than fluid.

In the knee we test for "floating of the patella" over an effusion by surrounding the joint with the hands, which are pressed slightly toward each other to limit the escape of fluid in either direction, and then *suddenly* making quick pressure on the patella with one finger. If we feel or hear the patella knock against the bone below and rebound as we release the pressure, fluid in abnormal quantity is present.

(*c*) *Irregularities of contour* are easily recognized, provided the normal contour is familiar.

(*d*) *Bony outgrowths* may be obvious (as in Heberden's nodes), but if within the joint they may be recognized only by the *sudden arrest of* *an otherwise free joint motion* at a certain point. In many cases radioscopy is necessary.

(*e*) *Gouty tophi* are identified positively by transferring a minute piece to a glass slide, teasing it in a drop of water, covering with a cover glass, and examining with a high-power dry lens and a partly closed diaphragm. The sodium biurate crystals are characteristic.

Fluid or semi-fluid exudates in joints may fill up and smooth out the natural depressions around the joint, or, if the exudate is large, may bulge the joint pockets; in the knee-joint four eminences may take the place of the natural depressions, two above and two below the patella.

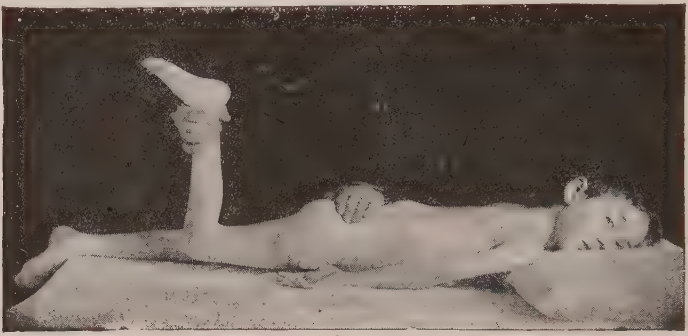

Fig. 264.—Testing for Psoas Spasm. (Bradford and Lovett.)

(*f*) *Limitations of motion due to muscular spasm* are seen with especial frequency in tuberculous joint disease, but may occur in almost any form of joint trouble, particularly in the larger joints.

(1) *Hip-joint*, two forms of spasm are important: (1) That which is due to irritation of the psoas alone (*psoas spasm*); (2) that in which *all the muscles* moving the joint are more or less contracted.

In pure *psoas spasm* the thigh is usually somewhat flexed on the trunk, though this may be concealed by forward bending of the latter. Very slight degrees of psoas spasm may be appreciable only when, with the patient lying on his face, we attempt hyperextension (see Fig. 264).

The other motions of the hip—rotation, adduction, abduction, and flexion—are not impeded.

General spasm of the hip muscles is tested with the patient on the back upon a table or bed (a child may be tested on its mother's lap) and the leg flexed to a right angle, both at the knee and at the hip.

Using the sound leg as a standard of comparison, we may then draw the knee away from the middle line (abduction), toward and past the middle line (adduction), and toward the patient's chest (flexion). Rotation is tested by holding the knee still and moving the foot away from the median line of the body or toward and across it.

(2) *Spinal column.* Muscular spasm of the muscles guarding motion in the vertebral joints can be tested by watching the body attitude (a stiff, "military" carriage in most cases), and by efforts to bend the spine forward, backward, and to the sides.

In most cases we can make out limitation of these motions by asking the patient to stand with knees and hips stiff and then bend his trunk (of course, naked) as far as he can in each of the four directions. If we are familiar with the average range of motility in each direction and at the different ages, this test is usually easy and rapid. Backward bending is the least satisfactory, and in doubtful cases the patient should be on his face, while the physician, standing above him, lifts the whole body by the feet (see Fig. 265).

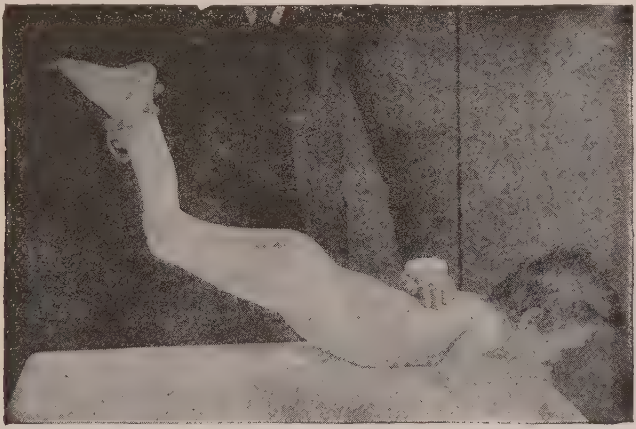

FIG. 265.—Rigidity of Spine in Pott's Disease.

(3) In the joints of the shoulder, knee, elbow, wrist, ankle, toes, and fingers, there is usually no difficulty in testing for muscular spasm, and no special directions are needed.

To distinguish muscular spasm from bony outgrowth as a cause of limited joint motion, we should notice that bony outgrowths (*e.g.*, in the hip) allow perfectly free motion up to a certain point; then motion is arrested suddenly, completely, and without great pain. Muscular spasm, on the contrary, checks motion a little from the outset, the resistance and pain *gradually* increasing until our efforts are arrested at some point, vaguely determined by our strength and hard-heartedness and by the patient's ability to bear the pain.

Motions limited by capsular thickening and adhesions are not, as a rule, so painful after the first limbering-up process is over. There is no sudden arrest after a space of free mobility, but motion is limited from the first and usually in all directions, though the muscles around the joint are not rigid. The possibility of more or less limbering-out after active exercise (or passive motion) distinguishes this type of limitation.

In true ankylosis there is no motility whatever.

(*g*) *Excessive motion* in a joint is recognized simply by contrast with the limits furnished us by our knowledge of anatomy and of the physiology of joint motion at different ages. When the bone and cartilage appear normal or are not grossly injured, we call the excessive motility of the joint a *subluxation*, but excessive motility may also be due (as in Charcot's joint) to destruction of bone and other essentials of the joint.

(*h*) To detect *crepitus and creaking* we simply rest one hand on the suspected joint, and with the other put it through its normal motions, while the patient remains passive.

(*i*) Most *free joint bodies* are not palpable externally, and are recognized only by their symptoms, by the *x*-ray, and by operation.

(*j*) *Shortening of a limb* as evidence of joint lesions is tested by careful measurements. The vast majority of such measurements are made with reference to the *hip-joint*. The tip of each anterior superior iliac spine is marked with a skin-pencil, and likewise the tip of each inner malleolus. Then, with the patient lying at full length on a flat table, the distance from anterior superior spine to inner malleolus is measured with a tape on each side.

The method of obtaining the other data tabulated on page 480 needs no explanation, except the radioscopic technique—a subject which I am not competent to discuss.

II. JOINT DISEASES

I shall use the classification proposed by Goldthwait and divide joint diseases as follows:

1. Infectious arthritis: (*a*) Tuberculosis. (*b*) Other infections.
2. Atrophic arthritis: (*a*) Primary. (*b*) Secondary to organic nerve lesions (Charcot's joint).

3. Hypertrophic arthritis.
4. Gouty arthritis.
5. Hæmophilic arthritis.

1. *Infectious Arthritis*

Under *infectious arthritis* are included all varieties of articular "rheumatism" and the joint troubles symptomatic of gonorrhœa, of streptococcus infections (including scarlet fever), influenza, syphilis, typhoid, and other fevers. As tuberculosis is an infection we must include it in this group, although the disease begins usually as an osteitis and involves the joint secondarily by extension.

I. *Tuberculous Arthritis.*—The characteristics of joint tuberculosis are:

(*a*) *Slow progress*, with gradual enlargement and disabling of the joint.

(*b*) *Muscular spasm*, especially in disease of the hip or vertebræ.

(*c*) Evidences of *low-grade inflammation* (moderate heat, swelling, pain, and tenderness).

(*d*) *Abscess* and *sinus formation.*

(*e*) *Malpositions* (*e.g.*, shortening of one leg in hip-joint disease, angular backward projection in spinal disease, subluxations in the knee-joint).

(*f*) *Bone necrosis*, as shown by *x*-ray.

The order of frequency in the different joints is as follows: spine, hip, knee, wrist, shoulder (tuberculous dactylitis is described on page 56).

In the deep-seated *hip-joint*, diagnosis has to depend largely on *shortening* and on the presence of *limitation of all the hip motions* by muscular spasm (see above, page 482), unless the disease is of long standing and manifests itself by *abscesses* burrowing to the surface. Usually these abscesses point in the upper anterior thigh, but they may open behind the great trochanter, below the gluteus maximus, or at any point in the vicinity of the hip.

Besides muscular spasm, shortening, and abscess formation, we get some aid from the general and vague joint symptoms present in this as in many other joint lesions. Such are enlargement (felt as thickening about the great trochanter), muscular atrophy, pain, tenderness, and crepitus.

In *spinal tuberculosis* (*Pott's disease*) the distortion of the bones with formation of a knuckle in the back is often obvious and practically diagnostic. In other cases we depend on muscular spasm or abscess formation. The muscular spasm gives a stiff back and often psoas contraction (see below). The abscess is peculiar, in that it usually works along in the sheath of the psoas and points in the groin below Poupart's ligament (see Fig. 239); less often it appears in the back or in the gluteal region, and rarely it may invade almost any part of the body, lung, gullet, gut, peritoneum, rectum, hip-joint, etc.).

Psoas spasm, which is common both in hip and spinal tuberculosis, is by no means peculiar to these diseases, and it is worth remembering that it may be due to various other lesions, such as:

(*a*) Hypertrophic arthritis of the spine.
(*b*) Appendix abscess.
(*c*) Perinephritic abscess.

In the peripheral joints (shoulder, elbow, wrist, finger, knee, ankle) the diagnosis of tuberculosis rests on the chronic enlargement and disability, with abscess and sinus formation.

Hysterical or acute traumatic lesions (with or without neurosis) may present symptoms and signs identical with those of tuberculosis. Decision is aided most by: (*a*) The lapse of time and the effects of treatment. (*b*) *X*-ray examination. (*c*) The predominance in functional and traumatic cases of pain and tenderness rather than muscular spasm or malposition.

II. *Acute Infectious Arthritis.*—All varieties are distinguished from the other types of arthritis by: (*a*) *The absence of* any marked *bone lesions*[1] in most cases. (*b*) The *tendency to recovery* in the great majority of cases.

The milder forms, whose cause is unknown, we have hitherto designated as "*rheumatism.*" The others are distinguished as gonorrhœal, pneumococcic, syphilitic, influenzal, dysenteric, etc., according to the organism producing them.

Between this group and those known as "rheumatism," there is no clear pathologic distinction. Mild infection with streptococci may leave a sound joint. Virulent infections may lead to crippling through fibrous adhesions. On the other hand, arthrtitis of "rheumatic" (*i.e.*, of streptococcic) origin may end in suppuration, crippling the joint with adhesions, though in most cases it leaves a sound joint.

All the members of the infectous group of joint lesions present the *local signs of inflammation* and the *constitutional signs of infection.* All may be complicated by endocarditis, but in those of streptococcic origin ("rheumatic") this complication is especially common. There is no hypertrophy, bone destruction,[1] sinus formation, or marked irregularities of contour. A general enlargement (more or less spindle shaped, owing to periarticular thickening and muscular atrophy) is

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[1] Exceptionally, virulent infections (especially those due to pneumococci or gonococci) may destroy cartilage and bone and end in true bony ankylosis.

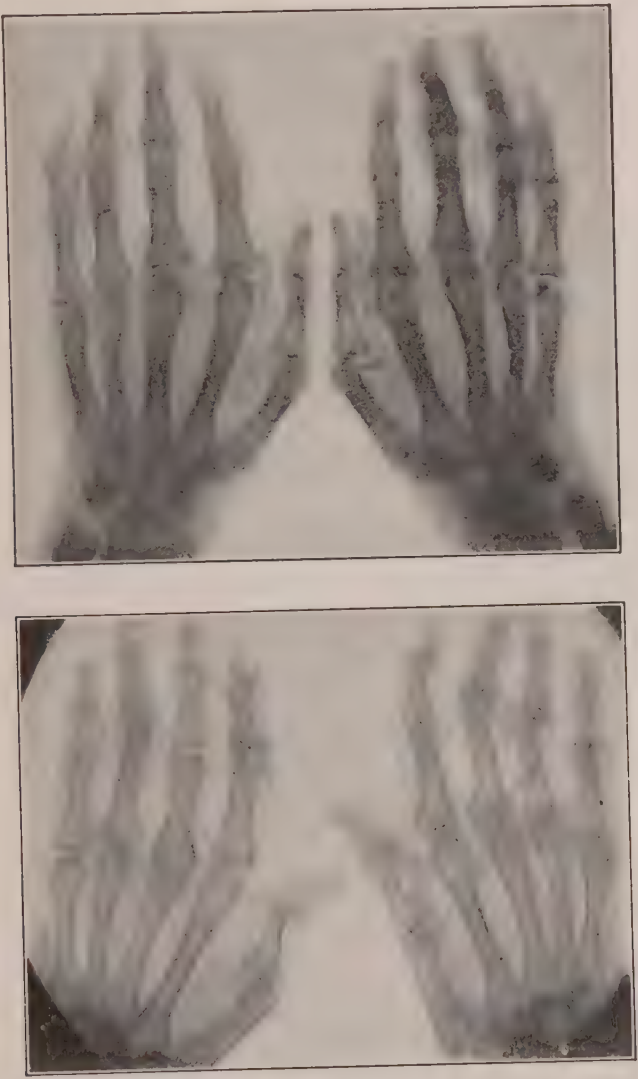

FIG. 266.—*X*-ray, showing Hands in Atrophic Arthritis.

the rule. The joint motions are limited chiefly by pain and effusion; muscular spasm is not prominent.

One or many large or small joints may be affected in any of the varieties of infectious arthrtitis, though the gonorrhœal virus is apt to lodge in few joints (oftenest the knee or ankle) and the "rheumatic" virus in many joints, while the typhoid poison has a predilection for the spine.

2. *Atrophic Arthritis*

Two types must be recognized: (*a*) A monarticular form, secondary usually to tabes or syringomyelia ("Charcot's joint," "neuropathic joint"), and other diseases of the spinal cord. (*b*) A polyarticular primary form ("rheumatoid arthritis" or "ankylosing arthritis").

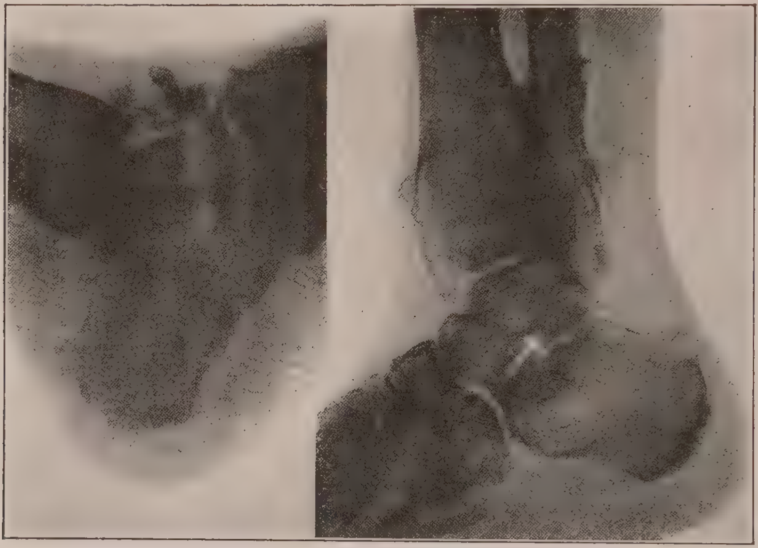

FIG. 267.—*a*, Charcot's Joint with Loose Bodies; *b*, Pulmonary Osteo-arthropathy. (v. Ziemssen's Atlas.)

In both, the distinguishing characteristic is atrophy and destruction of cartilage, bone, and joint membranes—a process which in the early stages can be identified only by the x-ray (see Fig. 266). Later the disintegration of the joint is usually evident, and is followed by notably painless distortions, contractures, and ankylosis.

(*a*) The *monarticular form* is generally easy to recognize on account of its rapid, painless course, with semifluctuant swelling, secondary to a well-marked cord lesion, such as locomotor ataxia. A large joint is almost always affected, oftenest the knee, less often the hip, shoulder, or elbow. The joint shows abnormal mobility and the bones can often be felt to grate (see Fig. 267).

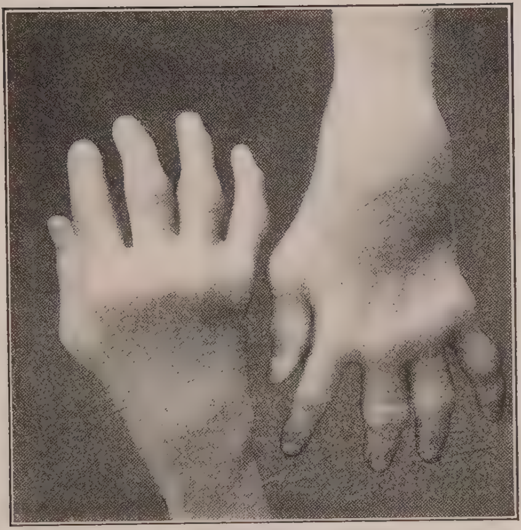

Fig. 268.—Atrophic Atrophic. Early stage.

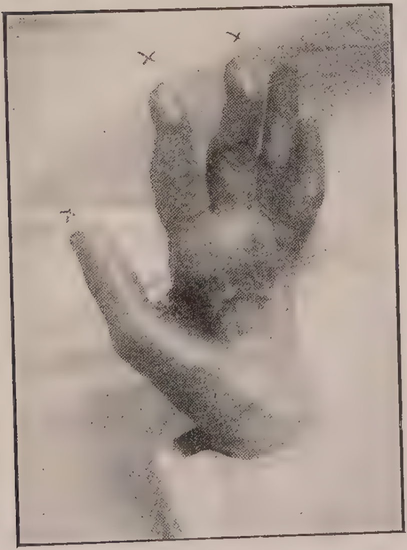

Fig. 269.—Atrophic Arthritis. (Goldthwait.)

(b) The *primary polyarticular form* usually begins in the fingers, and is very apt to occur *symmetrically*, *i.e.*, in corresponding joints of both hands at the same time (see Fig. 268). The joints are enlarged, boggy, spindle shaped (owing to the rapid atrophy of the interossei), often abnormally white, apparently fluctuant, and show trophic skin lesions (glossy skin, sweating, mottling) (see Fig. 269). The terminal finger-joints are rarely swollen. Late in the course of the disease a ring of constriction often marks the line of articulation (see Fig. 270). Pain is not severe until motion is attempted or unless the joint is jarred and stirred up by some traumatism.

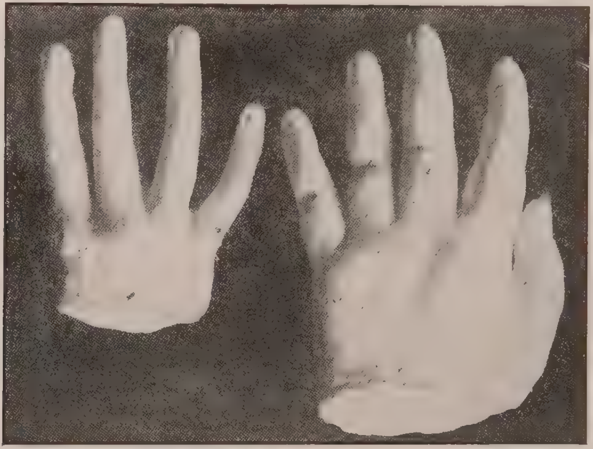

FIG. 270.—Atrophic Arthritis. Late stage with constriction ring at the joint line. (Goldthwait.)

The changes progress slowly and attack new and larger joints, moving centrally from the periphery. At any stage the process may become arrested, but usually not until *ankylosis* or *contractures* have occurred in one or many joints. Some of the "ossified men" of dime museums are in the ankylosed stage of this terrible malady. Flexion of fingers with hyperextension of the terminal joints, and deflection to the ulnar side are common deformities.

3. *Hypertrophic Arthritis*

This is degenerative type of disease in which osteophytic spurs are the distinguishing feature. It occurs mostly in elderly persons. The new bone is deposited round the edges of the articular cartilage, forming an irregular ring ("ring bone" in horses) or "lip" near the joint. The attachments of the ligaments (*e.g.*, the anterior lateral ligament of the spine, or the cotyloid ligament in the hip-joint) furnish another favorite site for the bony deposits. There is no ankylosis and motion is limited only by the collision of bony spurs in joint margins.

(*a*) In the terminal finger-joints ("*Heberden's nodes*") the process may remain for years without extending to any other articulation and without producing any discomfort (Figs. 54 and 271).

(*b*) The disease may be limited to the hip-joint ("morbus coxæ senilis") or to any other single joint, producing purely mechanical disturbances by limitation of motion. There is no considerable muscular spasm, and motion is quite free up to a certain point, at which it is suddenly "locked" by the interference of the bony outgrowths. The situation, size, and shape of these outgrowths can be shown, as a rule, by the *x*-ray alone. Pain and swelling are slight or absent, unless traumatism (internal or external) stirs up the joint and produces a synovitis. The chief complaint is of *stiffness*.

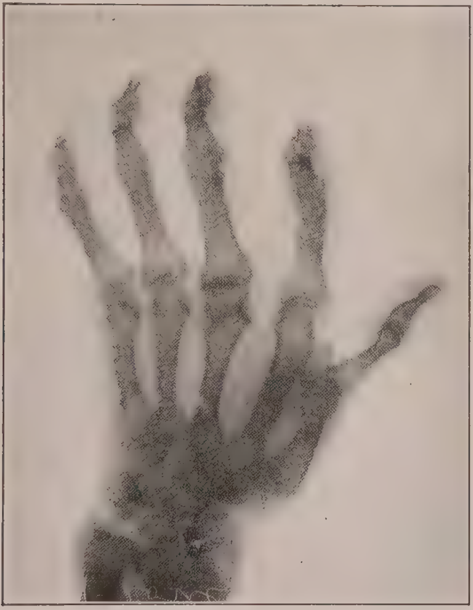

FIG. 271.—Hypertrophic Arthritis with Heberden's Nodes.

(*c*) Several joints may be affected, and there may result much pain because nerves pass through or over the new-formed bone and are compressed by it. This form is most often seen in the spine ("spondylitis deformans," "osteoarthritis"), where a portion of the front and side of the vertebral column is "plastered over" with new-formed bone (see Fig. 272), which later invades the intervertebral cartilage and produces (see Fig. 273) finally either a straight "ramrod" spine or a forward curved spine.

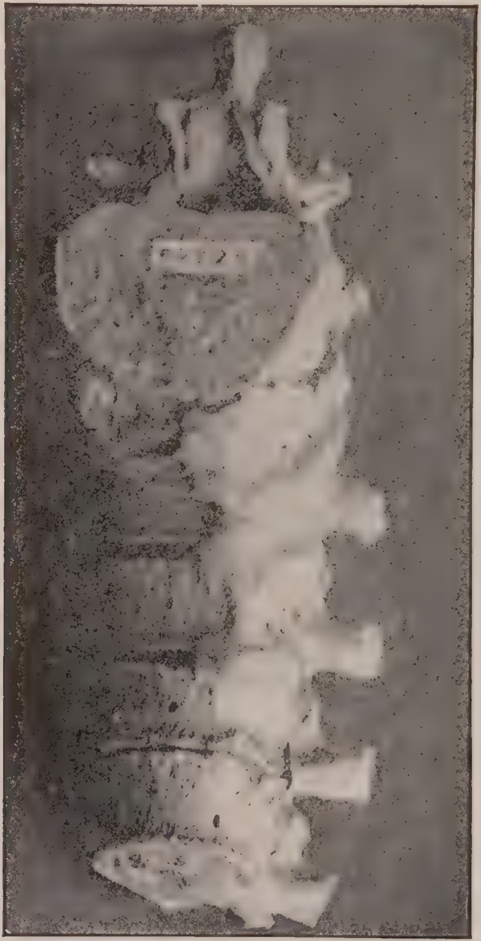

FIG. 272.—Hypertrophic Arthritis of Spine. (Goldthwait.)

Non-tuberculous disease of the sacro iliac joint has already been referred to on page 59.

In the early stages the disease is recognized by:

(*a*) *Nerve pain*, running round the body or down the legs,[1] as the intercostal and spinal nerves are pressed on.

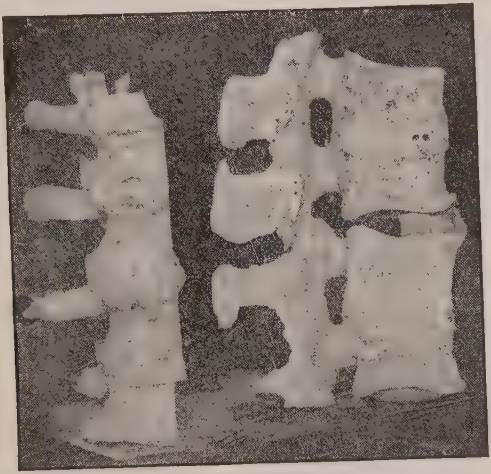

FIG. 273.—Hypertrophic Arthritis (Spine) of Spine with Ankylosis. (Goldthwait.)

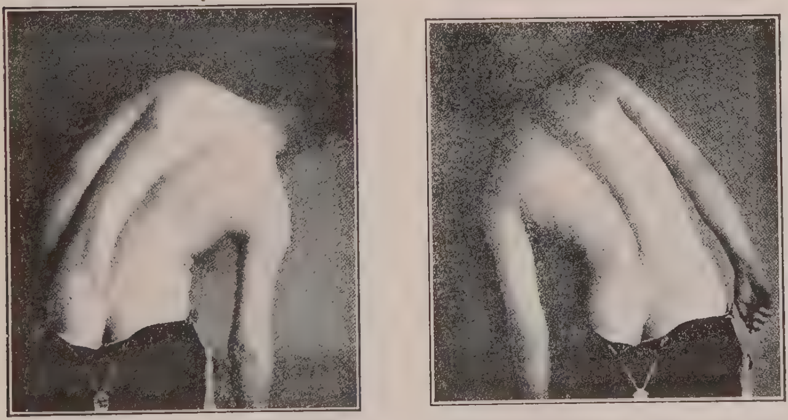

FIG. 274.—Showing Normal Flexibility of Spine. (Goldthwait.)

(*b*) *Limitation of Motion.*—The process is usually unilateral, wholly or predominantly; hence the patient can usually bend much better to one side (see Figs. 274 and 275) than to the other. Motion is also more or less limited in other directions, but forward bending is fairly well performed as a rule, in sharp contrast with "lumbago," which renders forward bending and the subsequent recovery almost impossible.

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[1] Many neuralgias and sciaticas are due to this disease.

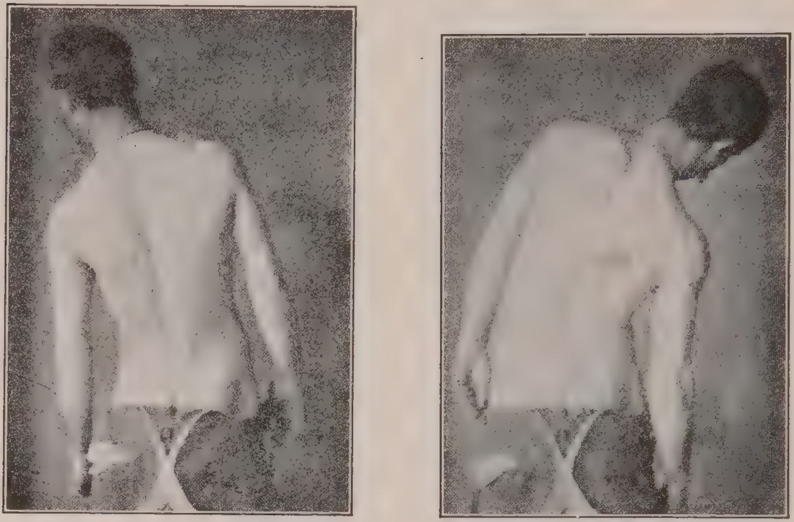

FIG. 275.—Hypertrophic Arthritis of Spine. Motion to left limited. (Goldthwait.)

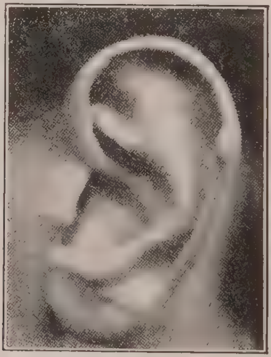

FIG. 276.—Gouty Tophus in the Ear.

(*c*) *Coughing or sneezing often gives great pain*, probably because the costo-vertebral joints are involved in the new growth; if ankylosis of these joints occurs later the respiratory movements of the chest are interfered with.

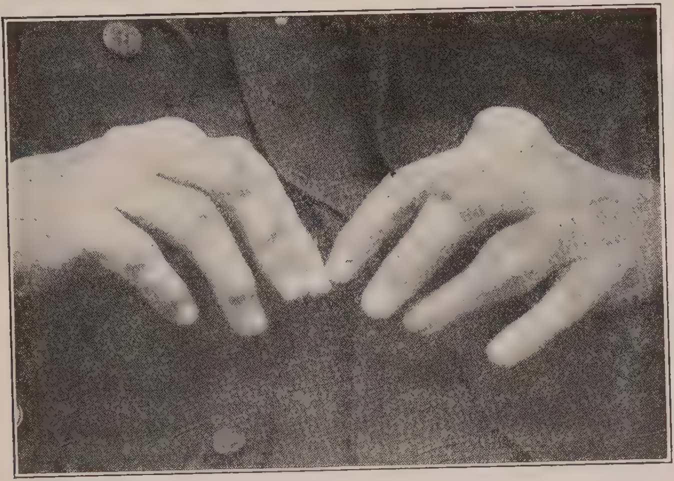

FIG. 277.—Gouty Arthritis.

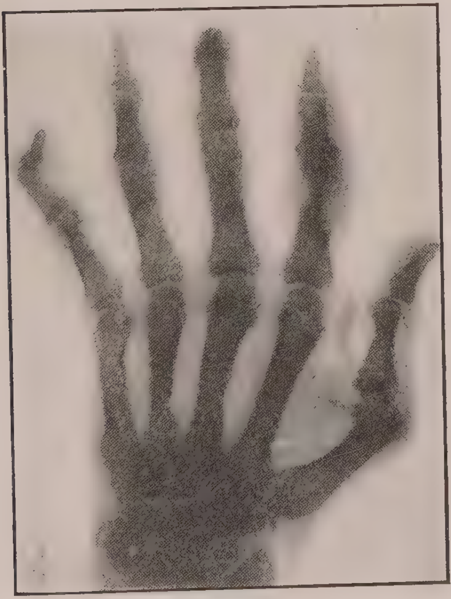

FIG. 278.—X-ray of Hand in Gouty Arthritis. (Goldthwait.)

4. *Gouty Arthritis*

The deposits of urate of sodium in the soft structures around the joint are, like those in the ear (see Fig. 276), close beneath the skin or perforate it, and hence are recognizable (as above explained) by microscopic examination.

They somewhat resemble the nodes of hypertrophic arthritis, but are not attached to the bone and can be moved about in the soft structures over it. *X*-ray examination shows that there is often considerable destruction of bone in the vicinity of the tophi (see Figs. 277 and 278).

5. *Hæmophilic Arthritis*

A chronic stiffening and enlargement of the joint resembling in many respects the joint of hypertrophic arthritis, but often accompanied by the formation of fibrous adhesions, ensures in some cases of hæmophilia, presumably as a result of frequent hemorrhages and serous oozings on the joint. The diagnosis depends on the evidence of hæmophilia, the youth of the patient, and the absence of infection as a causative factor.

6. *Relative Frequency of the Various Joint Lesions*[1]

The following table was prepared by Dr. Vickery[2] from the records of the Massachusetts General Hospital (1893–1903):

Infectious arthritis.	Acute rheumatic arthritis	591	873
	Subacute rheumatic arthritis	193	
	Gonorrhœal arthritis	86	
	Typhoid arthritis (spine)	3	
Hypertrophic and atrophic arthritis			43
Gout			9

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[1] Chronic villous arthritis ("dry joint") is a purely local process and therefore receives no further mention.

[2] *Boston Med. and Surg. Jour.*, November 17, 1904.

CHAPTER XVIII

THE NERVOUS SYSTEM

I. Examination of the Nervous System

The outlines of neurological diagnosis depend on knowledge of:

I. Disturbances of motion.

II. Disturbances of sensation.

III. Disturbances of reflexes (including sphincteric and sexual reflexes).

IV. Disturbances of electrical excitability.

V. Disturbances of speech and handwriting.

VI. Disturbances of nutrition ("trophic").

VII. Psychic disorders.

VIII. Changes in the spinal fluid.

I shall attempt no *topical* diagnosis of nerve lesions, no diagnosis, that is, depending on memorizing the brain areas, cord levels, or skin-and-muscle areas corresponding to particular nerve lesions. The general practitioner for whom this book is intended will not attempt to carry such points in his head, but will refer to specialists or special text-books when the case confronts him. The general methods most often employed are all that I attempt to describe.

1. *Disorders of Motion*

1. Gaits.
2. Paralyses.
3. Spasms and tremors.
4. Ataxia.

1. *Gaits.*—The most important gaits are:

(*a*) The *spastic*

(*b*) The *ataxic.*

(*c*) The *gait* of *paralysis agitans.*

(*d*) The *toe-drop gait*

(*e*) The *gait of simple weakness.*

With the *spastic gait* there is rigidity of the legs, making it difficult to lift the feet; hence the patient scuffs along, usually with bent knees and as if his feet were fastened to the ground.[1]

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[1] The *cross-legged gait* is a spastic gait in which the adductors of the thighs are so contracted that the feet tend to be crossed. This gait is oftenest seen in the *congenital spastic paralysis.*

The *ataxia gait* is difficult to describe. The patient is not muscularly weak, but does not know where his feet are or where the ground is; hence he flounders and throws his feet about irregularly.

The *gait of paralysis agitans* is an exaggeration of the old man's gait, such as we often see on the stage. The whole body is bent forward and rigid (see Fig. 279), and, if progress is accelerated by a push given from behind, the patient may be unable to stop himself.

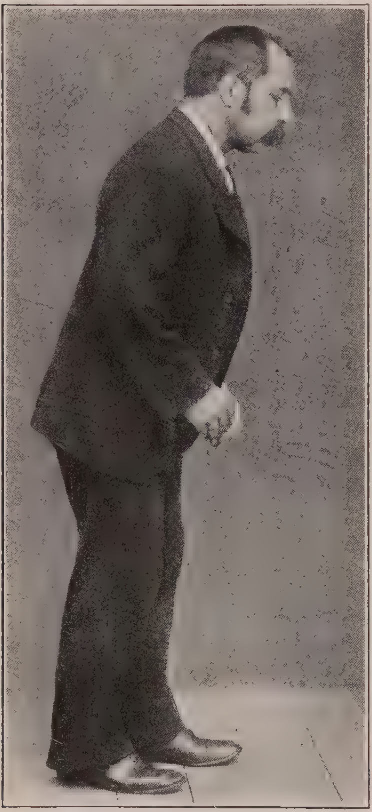

FIG. 279.—Attitude Characteristic of Paralysis Agitans.

In the *toe-drop gait* the foot is raised high and slapped down upon the ground with a flail-like motion.

2. *Paralysis or Paresis.*—No detailed account can be given here of the method of testing individual muscles for loss or impairment of power. In general, a knowledge of the origins and attachments of muscles enables us to work out for ourselves a series of tests that will bring any desired group into contraction. It is convenient to class paralyses according to their origin as follows:

(*a*) *Brain paralysis:* usually *hemiplegia* (arm and leg on same side, with or without the face).

(*b*) *Cord paralysis:* usually *paraplegia* (both legs, rarely both arms) or *monoplegia* (one extremity).

(*c*) *Cranial nerve paralysis:* usually *one or more eye muscles.*

(*d*) *Peripheral nerve paralysis: special muscle groups*, oftenest the extensors of the wrist or foot, the shoulder muscles, and those supplied by the facial nerve.

(*e*) *Hysterical paralysis:* no strict anatomical distribution, oftenest monoplegia (one extremity).

Peripheral nerve paralyses are especially apt to be accompanied by sensory symptoms, electrical changes, and wasting. Brain paralyses have relatively few sensory symptoms (sometimes paræsthesiæ, see below, page 501) and relatively slight wasting. Mental changes, coma, or convulsions often precede or follow them. Cord paralyses may or may not show these associations, but are often accompanied by disorders of the bladder and rectum.

3. *Spasm, Tremor, and Fibrillary Twitching.*—(*a*) Spasm means involuntary muscular contraction. The familiar "cramp" is a good example of the type of spasm known as *tonic spasm.* In contrast with this is the *clonic spasm*, in which flexors and extensors contract alternately to produce a motion like that of our forearm when we shake up a fluid in a test tube, or like the ankle clonus (see below).

Spasms may be general or local, *i.e.*, involve few or many muscles. In strychnine poisoning the whole body may be thrown into rigidity or *general tonic spasm.* At the beginning of an epileptic seizure the body stiffens out (tonic spasm), then becomes "convulsed" (*general clonic spasm*). *Local tonic* spasm is exemplified in the ordinary "*cramp.*" The spastic gait, above described, is another common example of tonic spasm limited mainly to one group of muscles. The *contractures* which so often affect the sound muscles in a partially paralyzed limb (see above, page 482) are also examples of *local tonic spasms.*

Athetosis, a special variety of local tonic spasm, has been described on page 47.

Local clonic spasm is not common. It may be due to irritation of a small portion of the cerebral cortex by various lesions ("Jacksonian epilepsy"), and sometimes precedes or alternates with the general spasms of ordinary epilepsy. It also occurs in hysteria.

Artificially a momentary or prolonged clonic spasm of the foot muscles is often produced in testing for the *ankle clonus* (see below, page 504).

(*b*) *Tremor* may be defined as a *clonic spasm of short excursion.* Its causes and varieties have already been discussed (see page 46).

(*c*) *Fibrillary twitchings* means the brief repeated contraction of small bundles of muscle fibres. It is seen in patients who are cold or nervous, in many debilitated and neurasthenic conditions and often in muscles affected by *progressive muscular atrophy.*

(*d*) *Choreic* and *choreiform* movements occur in true chorea, in encephalitis lethargica, and after hemiplegia.

4. *Ataxia.*—Inco-ordination of the various muscles which normally act together to produce a well-directed movement is called *ataxia.* All young infants exhibit ataxia in their more or less unsuccessful grasping movements. Alcoholic intoxication often produces typical ataxia,

and it is also exemplified in the *gait of tabes dorsalis.* There is no lack of muscular contraction—often too much—but it is disorderly and ill-directed.

Deficiency in the power to balance in standing or walking is perhaps the commonest type of ataxia, and may be due not only to the causes just mentioned, but also to cerebellar disease and ear disease. In these types there is often a tendency to stagger in one particular direction, *e.g.*, to the right, and the ataxia is associated with vertigo and with other evidences of brain tumor or of ear disease.

In tabes dorsalis and other diseases we test the power to balance by asking the patient to bring his feet together (toe to toe and heel to heel) and to close his eyes. If he is unable to preserve his balance his failure is known as "*Romberg's sign.*"

2. *Disorders of Sensation*

The following are the most important types:

1. *Anæsthesia* (or insensibility to pain, to touch, to heat and cold, and to muscle sensation).
2. *Hyperæsthesia* (or *oversensitiveness*).
3. *Paræsthesia* (abnormal, false, or disordered sensation).
4. *Pain.*
5. *Disorders of special sense.*

These disturbances may all be seen in different stages or types of lesions of the spinal cord or peripheral nerves. They are less common in brain lesions.

1. Tests of *anæsthesia* are time-consuming and difficult, because we depend for our data on the patient's intelligent answer to the question, "Do you feel that?" As a rule, we cover the patient's eyes and then touch the suspected parts—first lightly, then more strongly—questioning him to see if he feels the touch, can judge the nature of the touching object (finger, pencil, pin), and tell where he is touched. A pin-prick is oftenest used to test pain sense, and test tubes filled, one with hot, one with cold water, are convenient for trying the temperature sense. Finally, we try whether the patient can recognize familiar objects placed in his hand and can tell the position in which you may put his arms or legs. Failure to make these discriminations is known as *astereognosis*, and occurs oftenest in brain lesions affecting the temporal lobes.

Dissociation of sensation—the preservation, for example, of sensations of touch with loss of those of pain and temperature—occurs oftenest in syringomyelia.

Delayed sensation and mistakes regarding the point touched in testing are commonest in tabes dorsalis, which disease presents a great variety of sensory disorders not here catalogued.

The distribution of anæsthesia depends, like the distribution of paralysis, on the lesion. *Hemianæsthesia* is seen oftenest in *hysteria* and *organic brain lesions*. Cord lesions, such as *transverse myelitis* or *compression of the cord*, usually produce anæsthesia in the area supplied by the spinal nerves below the lesion. *Peripheral nerve lesions* may produce anæsthesia of the skin areas supplied by the nerve in question.

Areas of *hysterical anæsthesia* (with hyperæsthesia and paræsthesia) usually do not correspond to the distribution of any set of nerves or centres, and are distinguished by this fact.

2. *Hyperæsthesia* is most often recognized as hyperæsthesia for pain (tenderness) or in the special senses (sensitiveness to light or noise). It is commonest in peripheral nerve lesions and in hysteria. The tests are the same as those for anæsthesia.

3. *Paræsthesia* is commonest in the form of the familiar prickling and tingling felt when one's arm or leg has "gone to sleep." Sensations as of crawling insects are not uncommon, the "hot feet" of many elderly persons (with arterio-sclerosis) and the "burning hands" of many washerwomen are other familiar examples.

Local paræsthesia is not uncommon in lesions of the cerebral cortex, and constitutes the preliminary "*aura*" with which many attacks of epilepsy are ushered in. Well-developed *tabes dorsalis* shows many curious or distressing varieties of paræsthesia, as do many other varieties of peripheral neuritis.

3. *Reflexes*

We may distinguish:

1. *Pupil reflexes.*
2. *Deep reflexes* (tendon reflexes).
3. *Superficial reflexes* (skin reflexes).
4. *Sphincteric reflexes.*
5. *Sexual reflexes.*

1. *Pupil reflexes* have been described on page 16.

2. *Tendon Reflexes.*—Among the most important of these is the *knee-jerk* (quadriceps tendon); less important are the *ankle-jerk* (Achilles tendon) and *ankle clonus*, the wrist, elbow, and jaw reflexes.

To test the knee-jerk many methods are used; the following seems to me the best: The patient sits with his knees flexed at a blunt angle.

The physician lays his left hand on the front of the thigh and strikes the tendon of the quadriceps, just below the patella, with the finger tips of the right hand or with a rubber hammer. The left hand feels the sudden contraction of the quadriceps whether the foot jerks or not. If no contraction is obtained we should try what is known as "*reënforcement* of the knee-jerk." The essence of this is concentration of the patient's attention on a voluntary muscular contraction in another part of the body. We may accomplish this by asking the patient to hook the fingers of his hands together, and at a given signal to give a quick pull upon them and then let go. The physician gives the signal (often the word "now") and strikes the patella tendon at the same moment.

The knee-jerk is often wanting or feeble in young infants and in pneumonia. It varies a great deal in persons of different temperament; in high-strung or oversensitive persons and in the Jewish race very lively knee-jerks are often seen without disease.

Absence of knee-jerk is oftenest found in:

(*a*) Peripheral neuritis (alcoholic, diphtheritic, lead, etc.).
(*b*) Tabes dorsalis.
(*c*) Anterior poliomyelitis (on the paralyzed side).
(*d*) In the deepest coma from any cause.
(*e*) In complete severing of the spinal cord.

Given a case without knee-jerks:

Neuritis is suggested by the history (alcohol), by the presence of marked sensory symptoms (pain, tenderness), and the absence of symptoms pointing to the brain or cord.

In *tabes* the Argyll-Robertson pupil, the disturbance of the sphincters and sexual power, the "lightning pains," here and there, the changes in the spinal fluid (see page 510), and later the ataxic gait are important confirmatory signs.

Anterior poliomyelitis presents a flaccid paralysis, usually of one extremity, coming on suddenly in a young child and wholly without sensory symptoms.

Comatose patients, if the coma is due to cerebral hemorrhage and is not of the profoundest type, often show increased knee-jerks on the paralyzed side; but in very profound unconsciousness all reflexes are lost.

Partial destruction of the cord often increases the reflexes, but total division usually abolishes them.

Increased knee-jerk is found in:

(*a*) Cerebral paralyses (infantile, apoplectic, dementia paralytica etc.).

(*b*) Spastic paraplegia and the amyotrophic forms of lateral sclerosis.

(*c*) Many cord lesions, localized above the lumbar enlargement (transverse of pressure myelitis).

(*d*) The earliest stages of peripheral neuritis.

(*e*) Multiple sclerosis.

(*f*) Some forms of chronic arthritis.

DIFFERENTIAL DIAGNOSIS of cases with increased knee-jerks:

Cerebral paralyses usualy manifest their place of origin by the presence of psychic symptoms (coma idiocy, dementia) and by convulsions. The paralysis is usually hemiplegic and involves no wasting beyond the *atrophy of disuse*.

Spastic paraplegia is readily recognized by the gait (see page 497) and the absence of marked sensory or sphincteric symptoms. Its pathology is not known. If marked wasting of the muscles ocurs it is termed "*amyotrophic lateral sclerosis*."

Transverse or diffuse cord lesions above the lumbar enlargement produce usually anæsthesia below the level of the lesion and relaxation of the sphincters. Such cases are often syphilitic.

The *earliest stages of peripheral neuritis* are usually recognizable, despite a lively knee-jerk, by the predominant sensory symptoms and the etiology.

Multiple sclerosis presents, in typical cases, intention tremor (see above, page 46), nystagmus (page 17), and staccato speech. In atypical cases diagnosis is difficult and cases are often mistaken for hysteria.

Almost any *chronic joint disease*, except tuberculosis, may be associated with increased reflexes. Diagnosis depends on the absence of other causes for the increase.

Other Deep Reflexes.—The *Achilles reflex* is best obtained by having the patient kneel on the seat of a well-padded chair, with his feet unsupported, while we strike the Achilles tendon. The significance of its absence or increase is practically the same as that just given for the knee-jerk, but, since it represents a slightly lower position in the spinal cord, it may be affected earlier than the knee-jerk in any cord disease which begins at the bottom of the cord and travels up. Thus in tabes I have known the Achilles reflex absent when the knee-jerk still persisted.

Ankle clonus occurs in spastic conditions of the legs or in any disease which increases the other leg reflexes. It is obtained by supporting the patient's leg in a state of such relaxation as can be obtained, then suddenly and quickly forcing the foot up as far as it will go toward the shin, and holding it in this position. A clonic spasm results, which in *true ankle clonus* persists as long as we choose to hold the foot in this position. *Spurious clonus* is obtained when only a few contractions occur, the muscle then relaxing. This spurious clonus can often be obtained in neurasthenic and hysterical states, and has not the significance of true clonus.

Kernig's sign is a reflex hypertension of the ham-string muscles, when we flex the thigh on the trunk at a right angle (as in the sitting position) and then try to extend and lower leg. Its motion is arrested about half way between the right angle and full extension.

This reflex is of some value in the diagnosis of meningitis, though allowance must be made for the stiffness of old age. The sign is by no means pathognomonic, but is of some confirmatory value.

The precise opposite of Kernig's sign, viz: a great *slackness of the hamstrings* (hypotonus) is often a valuable confirmatory sign in *tabes dorsalis*.

The *deep reflexes of the arms* (wrist, biceps, and triceps tendon) are obtained by snapping these tendons sharply with the finger. Decreased reflexes we can rarely perceive, as a rule they are *obtainable only when increased*. Such increase may occur in the diseases which increase leg reflexes; also in chronic joint troubles.

The *jaw-jerk* is obtained by asking the patient to let the lower jaw drop fully, placing a finger on the chin and percussing that finger as in percussion of the chest. It can be elicited only when increased.

3. *Superficial Reflexes.*—A "ticklish" person is one whose superficial reflexes (skin and muscles) are very lively. Among pathological reflexes of this type:

(*a*) The *Babinski reflex* is the most important. It is a modification or reverse of the normal reflex, which crumples up the toes toward the sole of the foot if the skin of the foot is tickled.

To obtain the Babinski reflex, bare the patients foot and draw the blunt end of a pencil along the inner side of the sole from heel to toe with moderate pressure. If the great toe cocks up toward the shin, Babinski's reflex is present. Sometimes several other toes spread laterally and follow the great toe. Squeezing the calf or drawing the finger along the outer tibial surface of various other ways to irritating the lower leg may also bring out the Babinski reflex (Gordon's sign, Oppenheim's sign, etc.).

This reflex is obtained on the affected side in hemiplegia and other lesions involving the upper motor neuron.

(*b*) The *cremasteric reflex* draws the testis tight up against the body (as after a cold bath) when the *skin and muscles* on the inner side of the thigh are gathered up and firmly grasped in the hand.

(*c*) The *abdominal and epigastric* "tickle reflexes" are excited by lightly and quickly stroking the skin of these parts with a pencil point or something of the sort.

The presence of cremasteric, abdominal, and epigastric reflexes indicates that the portion of the spinal cord in which they are represented (upper lumbar and lower dorsal regions) is functionally sound. The absence of these reflexes, however, signifies nothing, for in many healthy persons they cannot be excited.

(*d*) The reflex of *winking* excited by the ordinary stimuli signifies the approximately normal conductivity of the fifth and seventh nerves (trigeminal and facial).

4. *Sphincteric Reflexes.*—The sphincters of the bladder and rectum are kept closed in the normal adult by reflex contraction, normally of moderate degree, but excited by the presence of urine and fæces. If there is no awareness of fæces at the anus or of urine at the neck of the bladder, owing to destruction of the conducting nerves or spinal nerve-centres, involuntary urination and defecation occur.

This is the case in transverse, diffuse, or compression myelitis above the segment (fourth and fifth sacral) where the centres for bladder and rectum are represented;[1] also in tabes dorsalis, dementia paralytica, and less often in other chronic spinal-diseases. Peripheral neuritis rarely affects the sphincters.

In deep coma from any cause (epilepsy, cerebral hemorrhage) the sphincters may be relaxed, owing to the abolition of sensation.

5. *Sexual Power.*—Sexual power may be regarded as a reflex in the presence of a particular stimulus, and is diminished or lost in chronic cord diseases involving the first and second sacral segments (lumbar enlargement) or the nerves leading to them, *e.g.*, in tabes, some cases of myelitis and dementia paralytica, etc. Temporary increase of power may precede the diminution.

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[1] It must be remembered that these nerves *arise from the cord* at the level of the first lumbar vertebra, though they do not *issue from the spinal column* till the fourth and fifth sacral foramina are reached.

4. *Electrical Reactions*

In health, a sharp contraction occurs if a faradic current is applied to a nerve or over a muscle, and a similar contraction can be obtained with the galvanic current just when the circuit is closed or broken, but not when the current is passing.

In contrast with these conditions is the *reaction of degeneration.* When this is present we obtain no muscular twitching with the faradic current and none over the nerve with the galvanic; but *with the galvanic over the muscle a slow, worm-like contraction* occurs, and the response to the positive pole is as good as to the negative, or better, whereas normally there is far better response to the negative. This is the *complete* reaction of degeneration; in *partial* reactions of degeneration all the normal reactions may be present, but diminished in intensity.

Reaction of degeneration occurs in all diseases affecting the anterior motor horns of the cord or their prolongations downward in the peripheral nerves; for example, in anterior poliomyelitis, progressive muscular atrophy, transverse or pressure myelitis, and all severe forms of peripheral neuritis. In brain lesions this reaction rarely occurs.

In *prognosis* a reaction of degeneration persisting after six to twelve weeks is unfavorable for recovery of the use of the muscles in which it occurs. If reaction of degeneration is absent or partial from the start, the prognosis is for relatively speedy recovery, *i.e.*, in weeks rather than months.

5. *Speech and Handwriting*

Aphasia, the loss of the power to speak or understand speech, despite normal hearing and muscular powers, occurs in lesions affecting the third left frontal and first left temporal convolutions of the brain.[1]

The lesions producing aphasia may be permanent anatomical changes following hemorrhage or tumor, or they may be transitory, as in uræmia and migraine.

The power to write or read letters is lost (*agraphia*) when the angular and supramarginal convolutions are destroyed.

Degeneration of the handwriting, as compared with the standard of former years, is often a helpful bit of evidence in the diagnosis of *dementia paralytica*, but may occur temporarily in various fatigue states.

[1] In some left-handed persons the centres are on the right side of the brain.

6. *Trophic or Vasomotor Disorders*

Trophic lesions of the joints, muscles (atrophy), skin, and nails have already been exemplified (pages 490 and 58). They blend with, and are by some explained as the results of vascular changes (*vasomotor*). *Herpes labialis* ("cold sore") and *herpes zoster* ("shingles") certainly seem to give every evidence of being due to nutritive disorders in the ganglia and not to vascular changes. The *acute bedsores* which form in myelitis, the "*angioneurotic*" *local swellings* which appear here and there in certain persons, and the local syncope or asphyxia which sometimes lead to Raynaud's form of gangrene, seem to need both nerve and vessel changes to explain them.

In brain lesions these trophic and vasomotor changes are much rarer than in disease of the cord and peripheral nerves.

7. *The Examination of Psychic Functions*

The diagnosis of the mental factors of disease forms an important part of the study not only of neurology, but of all diseases wherever situated; but as it cannot be called physical diagnosis, it falls outside the scope of this book, except in so far as loss of consciousness, *coma*, may be considered under this heading.

(*a*) *Coma*

The causes of coma are nearly identical with the causes of convulsions. Almost every disease which causes the one may cause the other; hence all that is here said on the diagnisis of coma applies equally well to the diagnosis of convulsions. Either or both may result from:

1. Cerebral compression (skull fractures).
2. Apoplexy (including cerebral hemorrhage, embolism, and acute softening thrombosis).
3. Alcohol.
4. Epilepsy.
5. Cerebral concussion (stun).
6. Uræmia and hepatic toxæmia.
7. Diabetes.
8. Syncope (fainting).
9. Opium.
10. Hysteria.
11. Gas poisoning.

12. Sunstroke.
13. Stokes-Adams' syndrome.

Apoplexy is the probable diagnosis when an elderly person who has shown no previous signs of ill-health becomes suddenly and deeply comatose within a few seconds or minutes. If hemiplegia is present (with or without aphasia) and if we can exclude the other causes above mentioned, the probability of apoplexy is increased. To determine hemiplegia in a comatose patient, try the following tests:

(*a*) Lift the arm and then the leg, first on one side and then on the other, and let go. The supported member falls more limply on the paralyzed side.

(*b*) Pinch or prick the limbs alternately. The sound limb may be moved, while the other remains motionless. Pressure over the supraorbital notch may bring out a similar difference in the response of the two sides.

(*c*) Try the knee-jerks. On the paralyzed side the jerk may be increased.

(*d*) Try Babinski's reaction. It may be present on the paralyzed side or on both sides.

Uræmia.—The diagnosis between apoplexy and uræmia is sometimes impossible, since uræmia may produce hemiplegia, and the urine in the two conditions (as obtained by catheter) may be identical. In practically all cases, however, the uræmic patient has previously shown obvious signs of nephritis—œdema, headache and vomiting, hypertrophy, oliguria, or polyuria with albuminuria. "Acute uræmia" suddenly appearing in a person apparently healthy is almost always a false diagnosis. The cases turn out to be skull fracture, apoplexy, arterio-sclerosis, etc. Convulsions more often precede or follow the coma of uræmia than that of apoplexy. Retinal hemorrhages or albuminuric retinitis, if recognized by ophthalmoscopic examination, point to uræmia.

The hepatic toxæmia in which many cases of cirrhosis and acute yellow atrophy die is distinguishable from uræmia only if the previous history of the case is known to us and the signs of liver disease (ascites, jaundice, and enlarged spleen) are evident.

Diabetic coma is usually recognized with ease, because the evidences of advancing diabetes lead gradually up to it. Like uræmia and unlike apoplexy it very rarely appears "out of a clear sky." The emaciation of the patient, the sweetish odor of the breath, the presence of sugar, and especially the evidences of acetone and diacetic acid in the catheter-urine, are the essential factors in diagnosis. Dyspnœa ("air hunger") precedes the coma in about one-third of the cases.

Concussion (or stun) after a blow usually clears up in a few minutes and so presents no difficulty in diagnosis. If the coma lasts on for hours or days (as it sometimes does) the suspicion arises that we are dealing with

Compression. For this the evidences are: Focal symptoms, convulsions, slowing of the pulse, and signs of depressed fracture. To determine the latter fact may be impossible even with x-ray, since the inner table of the skull may be broken, while the outer is intact. The focal signs to be looked for are paralyses (ocular or peripheral).

Syncope (or fainting) is usually over in a few minutes and so betrays its nature, but it must not be forgotten that a slight convulsion may occur just as the patient comes out of coma. No suspicions of epilepsy need be aroused thereby, but if there have previously been signs of hysteria we may be in doubt whether the fainting fit is not of hysterical origin. The history of the case, the circumstances at the onset of the attack, and the presence or absence of hysterical behavior during it usually guide us aright.

Opium poisoning produces a coma from which the patient can usually be more or less aroused. Contracted pupils and slow respiration are the most characteristic signs. A laudanum bottle or a subcutaneous syringe found near the patient often assist the diagnosis.

Alcoholic coma is rarely complete. The patient can be aroused. The circumstances under which he is found, the odor of alcohol on the breath, the absence of paralysis, fever, small pupils, or urinary abnormalities are the main supports in diagnosis. There is no characteristic pulse and the pupils show no constant changes, though in many cases they are dilated.

Hysterical coma usually occurs in young women who have previously shown signs of hysteria. In falling they never hurt themselves. The eyelids are contracted, often tremulous, and when forcibly pulled open often expose eyeballs rolled up so that the whites alone are seen. The hands are apt to make grasping motions, and there are irregular, semipurposive movements of various parts of the body. A startling word may arouse the patient, but anæsthesia to pain (over one-half or all the body) is often complete.

Postepileptic coma is usually recognized with ease, because of the convulsions which precede it and which are usually known to have occurred at intervals before. The scar of previous falls may be found on the head.

Gas poisoning rarely presents any diagnostic difficulties, because the circumstances under which the patient is found make clear the cause of his condition. An odor of gas may hang about his breath for some hours.

Sunstroke is recognized by the state of the weather and the presence of a very high temperature (106°, 110°, 115° F., or even more). There is no other characteristic sign. This condition is to be distinguished from *heat exhaustion* in which there is no fever and no coma.

The *Stokes-Adams' Syndrome* (see above p. 114) produces coma and convulsions with a very slow radial pulse and a quicker venous pulse (visible or traceable polygraphically) in the neck.

8. *Examination of the Cerebro-Spinal Fluid*

In cerebro-spinal syphilis (including tabes and paresis), in lethargic encephalitis and in all types of meningitis, the character of the fluid obtained by lumbar puncture is of great diagnostic value. In all comatose and paralytic states it may be useful.

To obtain spinal fluid, a hollow needle is inserted between the spines of the vertebræ in the median line at the level of a line carried round the back from the crest of one iliac bone to the other. The needle is pushed straight on until fluid begins to flow through it. This usually occurs when the needle point is about two inches from the surface. If the spinal fluid is under tension it may spurt through the tube but usually it falls drop by drop into the test tube, which is held in position to receive it.

The most important tests are: 1. The cell count. 2. The differential cell count. 3. The Wassermann test. 4. The Lange colloidal gold test. 5. The quantitative test of sugar.

To *count the cells* one puts a drop of the well shaken fluid on the counting disc of the Thoma-Zeiss blood counter and proceeds exactly as in dealing with blood. Normal cerebro-spinal fluid contains from 1 to 10 (usually 1 to 5) cells per cm. In syphilis from 20 to 200 or more cells are often found. In meningitis and pneumonia there are often several hundred. In leucæmia the count rises into the thousands.

The *differential count* shows in most chronic inflammations of the canal, *i.e.* in tabes, paresis, and tuberculous meningitis, 90 to 100 per cent. of lymphocytes. In acute irritations such as epidemic meningitis and pneumonia the cells are largely polynuclear. In poliomyelitis the polynuclears range from 0 to 60 per cent. The rest are lymphocytes. There is also a considerable lymphocytic increase in lethargic encephalitis but in this disease an increase of the sugar in the spinal fluid is the most characteristic point, especially when this increase reaches as high as .07 to .09 per cent. (normal 0.4 to .06 per cent).

The *Wassermann reaction* is often positive in the spinal fluid when it is negative in the blood, *e.g.* in tabes. Its value is therefore obvious.

The *Lange gold test* depends on a color change produced by certain spinal fluids in a solution of colloidal gold. It is valuable chiefly in the recognition of dementia paralytica. Bacteriologic examination by culture or more often by cover.

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